A STUDY OF INCIDENCE AND PRESENTATION OF THYROIDITIS IN A GOITROUS PATIENT AT GRH MADURAI

DISSERTATION SUBMITTED FOR

M.S. DEGREE BRANCH I (GENERAL SURGERY)

MARCH – 2010

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI - TAMILNADU

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CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY OF INCIDENCE AND PRESENTATION OF THYROIDITIS IN A GOITROUS PATIENT"** submitted by **Dr. P. VIJAYAKUMAR** to the Faculty of Surgery, The Tamil Nadu Dr.M.G.R. Medical university, Chennai in partial fulfillment of the requirement for the award of M.S. Degree in Surgery is a bonafide work carried out by her during the period of May 2007 – Nov 2009 under my direct supervision and guidance.

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DECLARATION

I, Dr. P. VIJAYAKUMAR solemnly declare that the dissertation titled **"A STUDY OF INCIDENCE AND PRESENTATION OF THYROIDITIS IN A GOITROUS PATIENT"** has been prepared by me.

This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement for the award of Master of Surgery, **(Branch I) General Surgery** Degree Examination to be held in March 2010.

Place : Madurai

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DR. P.VIJAYA KUMAR

ACKNOWLEDGEMENT

It gives me great pleasure to acknowledge all those who guided, encouraged and supported me in the successful completion of my study.

I I express my gratitude to the Dean – In – Charge, **Dr.S.M. SIVAKUMAR, M.S.,** for allowing me to utilize the clinical materials for this study.

Foremost, I wish to express my sincere gratitude to **Prof. Dr. K.V.MAHESWARAN M.S.,** II Surgery Unit Chief, for his valuable support and continued guidance encouragement throughout the study.

I wish to thank my unit Assistant Professors **DR. SELVA CHIDAMBARAM M.S., D.R. CELINE FOUSTINA MARY, M.S. D.G.O., DR.ANTO FERNANDO M.S., DR. SUBBIAH CHANDRASEKAR M.S.,** for their valuable guidance and support.

In sincerely thank all Unit Cheifs and Assistant Professors of department of General Surgery for their co-operation and allowing me to do my study.

My sincere thanks to Prof. and Asst. Prof of dept of surgical endocrinology and medical endocrinology for their valuable guidance and motivation to complete this study.

I owe my sincere thanks to all my beloved patients for their kind co-operation in spite of their suffering in completing this study.

CONTENTS

INTRODUCTION

Thyroiditis is one of the commonest thyroid diseases, being particularly very common in south India (when compared to North India).

However, in view of the diagnostic limitations in thyroid swelling, the this condition is often missed and thyroidectomy is performed on which is not only unnecessary, but also harmful to the patients, which is not only unnecessary, but also harmful to the patients, as it will lead to early and inevitable hypothyroidism. The patients are also at risk of early and late complications of thyroidectomy e.g. recurrent laryngeal nerve injury, hypoparathyroidism, neck scar, etc.

Hence, the study is undertaken to elicit some of the diagnostic feature of the disease and thus to avoid surgery in these patients wherever possible.

AIM OF THE STUDY

- To study incidence and epidemiological aspects (age, sex) of thyroiditis in a goitrous patients.
- To find out the clinical features and presentation of thyroiditis in a goitrous patients.
- To find out the status of investigations (hematologic, biochemistry, immunology, histopathology and imaging studies)

STUDY METHOD

SETTING : Patients were selected from Medical Endocrinology Department and Surgical Endocrinology Department.

STUDY DESIGN: prospective observational study.

PERIOD: 2007 – 2009.

ETHICAL CLEARANCE The study was approved by the hospital ethical committee.

INFORMED CONSENT: Those who were willing to participate in the near1y after an informed consent were included in the study.

INCLUSION CRITERIA:

1. Clinical findings suggestive of thyroiditis, supported by investigations.
2. Final diagnosis affirmed by head of the department of Medical Endocrinology.
3. Only newly diagnosed patients were included in the study.

DIAGNOSTIC CRITERIA:

Since a histological evidence of Hashimoto's thyroiditis cannot be obstrainted practically routinely, the minimum requirement to diagnose a case as Hashimoto's thyroiditis was taken as 'positive AMA' (most of the books quote that AMA antibodies are present in 100% of the cases)

CASE SELECTION CRITERIA:

When history and clinical examination are suggestive of thyroiditis and it is supported by one or more of the three investigations (Thyroid function tests, Antimicrosomal antibody titer, FNAC).

NUMBER OF PATIENTS

Though the study started with- more than 300 patients, not all patients turned out for follow-up for complete investigations.

- Only those patients who underwent all investigations were included in the study. For ease of calculation, 234 number of patients were selected.
- The final diagnosis of all these 234 patients were confirmed by the head of the department of medical endocrinology & surgical endocrinology.

EXAMINATION METHOD:

- History from all patients were obtained by standard order of questioning as outlined in the Proforma given at end of this book.
- All the patients were clinically examined by a single examiner to avoid observer bias.
- FNAC smear was made by the same examiner and microscopic findings were noted by the same, with the help of a pathologist in the hospital. 23

gauge needle was used to aspirate cellular material and fixed immediately with 95% alcohol and stained H&E stain

- Antimicrosomal antibody titer estimation was done by Radioimmunometric assay.
- Thyoid hormonal assay was done by radioimmunoassay.
- ESR was estimated by Westegren’s method.

REVIEW OF LITERATURE

DEFINITION

Thyroiditis is defined as inflammatory disorder of the thyroid aland.

CLASSIFICATION OF THYROIDITIS

Acute (Suppurative)	-	Bacterial – staphylococcus, streptococcus
		Anaerobes, Fungal
	-	Drug, Radiation induced thyroditis
Subacute	-	Granulomatous (De Quervains)
	-	Lymphocytic (Silent, Painless)
	-	Post partum thyroiditis
Chronic	-	Hashimoto’s (Autoimmune)
	-	Reidel’s

HASHIMOTO'S THYROIDITIS

It was first described in Japan by Hawkin Hashimoto in 1912 and is the most well known of the immunological thyroid diseases. Hashimoto termed the disorder as 'Struma lymphomatosa'

Over the last 50 years, the number of new cases diagnosed per year in U.S. has risen exponentially. In Hashimoto's disease thyroid tissue damaged by immunological factors is replaced by lymphocytes, plasma cells and fibrosis. Antithyroid antibodies are present in the serum of patients with Hashimoto's disease being first described in 1957 by Donsach and associates.

Hashimoto's thyroiditis has been linked to other autoimmune disease including vitiligo, Addison's disease, Type I Diabetes Mellitus, Alopecia Sjogrens syndromes, Pernicious anemia, Lupus erythematoses, Rheumatoid arthritis, Myasthenia gravis and Idiopathic thrombocytopenic purpura.

Other predisposing conditions to autoimmune thyroiditis include Down syndrome, familial Alzhiemer' disease, and Turner's syndrome.

Hashimo to thyroiditis may rarely be associated with other endocrine disorders caused by the immune system. When Hashimoto's disease occurs

with adrenal insufficiency and type I Diabetes mellitus, the condition is called Type 2 polyglandular autoimmune syndrome (PGA II).

Less commonly, Hashimoto's disease occurs with hypoparathyroidism, adrenal insufficiency and fungal infections of the mouth and nails iii it condition called Type I polyglandular autoimmune syndrome (PGJA I)

ETIOLOGY AND PATHOGENESIS

A current popular theory for the pathogenesis of HT is that it is a disorder of impaired immune surveillance, with dysfunction of normal 'suppressor' T lymphocytes, allowing a clone of 'helper' T lymphocytes antigens, resulting in produce circulating to thyroid antibodies to thyroid destruction of thyroid cells.

Patients with Hashimoto's disease usually have detectable anti-thyroid antibodies at sometime in the course of their disease. The important cytotoxic effects of microsomal antibodies are increasingly recognized.

Lymphocytes from patients with Hashimoto's disease secrete lymphokines and undergo blast transformation when exposed to thyroid cells in vitro. The mechanism leading to anti thyroid antibodies formation and cell mediated immune reactivity has not been fully established.

Recently, however, it was suggested that HT may occur in absence of a systemic immune response. Baker et al. described a case of HT in which lymphocytes isolated directly from thyroid gland produced antibodies against Thyro globulin microsomal antigen, the thyroid cell membrane, and TSH, without there being serologic presence of thyroid autoantibodies. The authors suggested that intrathyroidal lymphocytes may be the source of thyroid directed antibodies. Thus, there may be more than one mechanism for the cellmediated immune response in HT.There appears to be a genetic predisposition for HT, as shown by its certain major histocompatibility complex antigens, such as HLA – B8 and HLA – DR5.

Only hypothesis is that a local virus may be responsible. Viruses cause production of interferons, and gamma-interferon is a known inducer of DR antigen expression. Thus, in patients with genetic predisposition, a viral infection may indirectly cause induction of thyrocyte lILA-DR expression, resulting in a cascade of antigen production, antibody formation rni1 coil destruction.

Another infectious agent, namely the gram-negative coccobacillus Yersinia enterocolitica is associated with positive thyroid autoantibodies and, conversely, antibodies to the Yersinia organism have been reported in between 50% and 90% of patients with autoimmune thyroid disease.

In addition to infectious agents, certain environmental agents, including iodides, have been implicated in the pathogenesis of HT. studies in genetically susceptible animals have shown that potassium iodide supplements lead to a significant increase in antibody titers in blood as well as lymphocytic proliferation of the thyroid. The apparent sensitivity to iodides agrees with the

clinical observation of an increasing prevalence of HT in industrialized, iodine – sufficient countries.

PATHOLOGY

The enlarged thyroid is pale, firm and pale yellow with a fine nodular surface. Adjacent lymh nodes may be enlarge.d histologically, there is diffuse infiltration of the gland by lymphocytes and plasma cells, with formation of lymphoid dollicles and germinal centers. The thyroid follicles are disrupted, and the follicular basement membrane is damaged. Some epithelial cells are enlarged and show a characteristic oxyphilic change in the cytoplasm (Askenazy cells).The disease is usually focal but gradually extends to involve the whole gland.

As the lymphocytic infiltration progresses, the thyroid tissue degenerates and may be replaced by fibrous tissue.

CLINICAL FEATURES

Middle aged women with grade I or II uniform enlargement of thyroid or obvious hypothyroidism are most likely to have Hashimoto's thyroiditis.

The most common presenting symptom is tightness in the throat, associated with a painless, nontender enlargement of the thyroid gland.

Gland is firm and mildly enlarged (usually grade I or II).

Enlargement is usually symmetrical, often granular on palpation.

The thyroid may be 2 to 3 times enlarged than normal size and as the lobules become more prominent, it may be finely nodular on palpation. In some the gland is frankly nodular rather than diffusely enlarged.

Pyramidal lobe may be enlarged.

Tenderness is uncommon.

Two different reports have been published- in one report, 100% of the patients had pain and tenderness over the gland; in another report, only 10% of the patients had tenderness over the gland. These observations stress the varied clinical presentations of HT.

Most patients are euthyroid when the diagnosis is made.

Approximately 20% of patients with Hashimoto's thyroiditis present with signs and symptoms of hypothyroidism; 5% of patients with hyperthyroidism. (Hashitoxicosis).

In the atrophic form of the disease, the thyroid is normal and the medical attention for symptoms of hypothyroidism

The prevalence of overt hypothyroidism increases with age.

Large goiters may be associated with pressure symptoms.

Compression of the trachea or a recurrent laryngeal nerve is rare.

Rapid enlargement of the gland should raise the suspicion of thyroid lymphoma or carcinoma.

Thyroid neoplasm is suspected clinically owing to asymmetry of the cervical lymphadenopathy, pressure symptoms, hoarseness, or enlargement of goiter despite adequate thyroid replacement. There is a relationship between thyroiditis and lymphoma. The tumour is rare but the risk of thyroid lymphoma is greatly increased in patients with Hashimoto's disease, in Comparison with the general population.

EPIDEMIOLOGY

It is the most common cause of goitrous hypothyroidism in adults and sporadic hypothyroidism in children.

World wide, the most common cause of hypothyroidism is iodine deficiency; however, Hashimoto's thyroiditis remains the most common cause of spontaneous hypothyroidism in areas of adequate iodine intake.

The incidence is 1-2 new cases per 1000 women per year.

Prevalance is 20cases per 1000 population.

The disease is 10 to 15 times more common in women than men.

The highest is in the age group of 20 to 40 years, peak incidence in men occurring 10 to 15 years later.

The annual risk of developing clinical hypothyroidism is about 4% when subclinical hypothyroidism is associated with positive TPO antibodies.

It-is rare in children; very rare in children below 5 years.

In adolescents, 40% of goiters are due autoimmune thyroiditis.

It may be familial.

Upto 50% of first degree relatives of patients with Hashimoto's thyroiditis have thyroid antibodies inherited as a dominant trait.

It is more common in areas of iodine excess.

It is common in certain populations such as the Japanese, probably as a consequence of genetic factors and chronic exposure to a high iodine containing diet.

DIAGNOSIS

LABATORATY INVESTIGATIONS FOR THYROID DISORDERS

HISTORY

Over the last several decades there has been an explosion in the number laboratory investigations available to the physicians for assessing status of a patient. Initially the physician was limited to such crude measurements as serum cholesterol level and basal metabolic rate in an attempt to establish either hypo or hyper thyroidism. These tests were nonspecific and offered little more than what was obtained through clinical evaluation of the patient. The rapid growth in thyroid function testing began in the mid l950s when the protein bound iodide (PBI) determination became widely available. It was the first labarotary test that could quantitate the thyroid status of a patient. The physician's ability to test the thyroid dysfunction improved with the introduction of competitive protein binding assay (1972) that more accurately measured of serum T4 levels. Estimates of free T4 levels as well as direct measures of biologically active free fraction of T4 recently have become available and play an integral role in modern thyroid function testing strategies. The major breakthrough, however in the arena of thyroid testing has been the development and evolution of the radio-immunoassay of thyrotropin(TSH), which serves as a readout of the endogenous free T4 levels in humans. Technological advancements in TSH assays have improved the ability to detect

the small changes in free T4 levels, allowing the physician to diagnose thyroid dysfunction earlier and to intervene before clinical disease is evident. Despite these laboratory methods, the doctor-patient relationship, a thorough history and a complete clinical examination remain critical in assessing the thyroid status of an individual.

APPROACH

Two fundamental questions need to be answered in the evaluation of a patient for possible underlying thyroid dysfunction, “what is the metabolic status of the patient?” and “what is the etiology of the disease?”

An excellent gauge of thyroid status is either the free T4 concentration or the serum TSH levels. It will be shown that TSH measured by a sensitive assay offers the most reliable means of determining thyroid status in a given individual. In addition, because most thyroid disease directly involves the thyroid gland itself and is mediated by autoimmune process, the physician must assess the susceptibility of the thyroid gland to autoimmune mediated disease. Currently the measurement of antibody (AMA), also known as an anti-thyroid peroxidase antibody (TPO) titer, is the most sensitive marker for the detection of underlying autoimmune thyroid dysfunction.

The following paragraphs present a cost – effective approach to detect thyroid dysfunction and susceptibility of the thyroid disease in any given patient. The current role and the limitations of a single serum TSR level as a marker of thyroid status are addressed as well. Finally, common clinical situation in which discrepancies in the thyroid function occur and when more extensive thyroid testing strategies are indicated are discussed.

Hashimoto's thyroiditis is nearly always associated with positive antithyroid antibodies and positive AMA can be detected in sera of approximately 90% or more of the eases. Antithyroglobulin antibodies (ATA) are less frequently positive, being present in between 20% and 50% of cases. From a clinical stand-point, obtaining the AMA test is sufficient in order to confirm the diagnosis of HT. Although the AMA test is less specific than the ATA test, it is significantly more sensitive. When antibody titers were measured in cytologically proven HT, significant AMA titers were present in 61 of 65 patients studies, whereas positive ATA titer were present in only 15 of 65 patients.

Clinically, the only tests necessary in patients in whom HT is suspected are the total serum T4, TSH level, and AMA test.

SERUM TSH ASSAYS

The most significant advance in the area of thyroid function testing has been the improvement in the accuracy of measuring low serum concentration (normal range: 0.40- 4.5 mU/I). When first developed in 1965 and then made available in mid-1970s, TSH radioimmunoassay could reliably detect TSH values at a level of 1 mU/I, limiting their utility to those situations in which serum TSH levels would be elevated, i.e. hypothyroidism. When the development of monoclonal antibodies to TSH and resultant immunometric assays improved by an order of magnitude to 0.1 mU/i. Termed ‘sensitive’ TSH assays, they allowed the measurement of TSH to be used for the first time in the diagnosis of hyperthyroidism.

The incorporation of chemoluminescent tags rather than radioisotopes in immunometric TSH assays had produced another order of magnitude of sensitivity, by which newer assays can routinely measure TSH concentrations at the 0.01 mU/l level. This has further improved the physician’s ability to reliably detect even milder form of thyroid hormone excess. Thus, assays capable of measuring TSH to the 0.1 mU/l level should be employed. Ideally, laboratories should offer assays that detect serum TSH at the 0.01 mU/l limit. These modern TSH methods should now be referred as second- generation (0.1mU/1 sensitivity limit) or third – generation (0.01 mU/1 sensitivity limit TSH assays.)

(0.1 mUll sensitivity limit) or third-generation (0.01 mUll sensitivity limit) TSH assays.

ANTIMICROSOMAL ANTIBODY TITERS

The immune state of the thyroid gland should be evaluated to determine the etiology of thyroid disease, because the majority of thyroid disease with altered thyroid function encountered in a clinical practice is autoimmune in origin (Graves' disease or Hashimoto's thyroiditis). Currently the most widely available and accurate test is the measurement of the AMA or Anti TPO titer. In the past, hemagglutination methods were employed for the measurements of AMA titers, but they were plagued by both insensitivity and nonspecificity. Recently, immunoassay techniques have been developed to accurately measure serum AMA titers (normal range : <0.5 all) whereby values are positive in greater than 90% of patients with underlying autoimmune thyroid disease. Further it has been demonstrated that upto 15% of the general population have measurable AMA titers, with positive titers being more common in women than in men as well as in the older population. Upto 5% per year of patients with a positive AMA titer and normal TSH value develop some form of thyroid dysfunction. Thus, AMA titers have both diagnostic and prognostic utility in the evaluation of the patient with suspected thyroid disease.

Tests for thyroglobulin and microsomal antibodies should be performed because the presence and the titer of these antibodies correlate with the severity and extent of the autoimmune process. The titer regarded as positive varies in different laboratories and with the particular method and the reagents hypothyroidism associated with a goiter but negative thyroid antibodies suggest use of goitrogen, a dishormonogenetic goiter or an endemic goiter. Thyroid peroxidase antibodies (formerly called antimitochondrial antibodies) are present in more than 90% of cases and thyroglobulin antibodies are present in about 50 % of the cases.

TIIYROID FUNCTION TESTS

Thyroid function tests should be performed to find out the hormone status. Patients are initially euthyroid but as the gland destruction progresses, hypothyroidism develops. Some patients in the early phase may develop hyperthyroidism. This is believed to be result from clones of lymphocytes that produce stimulatory anti TSH antibodies, creating a hyperthyroid status known as hashitoxicosis.

Because the autoimmune process gradually reduces thyroid function, there is a phase of compensation during which normal thyroid hormone levels

are maintained by a rise in TSH. Though some patients have minor symptoms, this state is called subclinical hypothyroidism. Later free T4 levels fall and TSH levels rise further; symptoms become more readily apparent at this stage (usually TSH > 10 mUlL), which is referred to as clinical hypothyroidism (overt hypothyroidism).

A normal TSH level excludes primary (but not secondary) hypothyroidism. If the TSH is elevated, a free T4 level is needed to confirm the presence of clinical hypothyroidism, but free T4 is inferior to TSH when a screening test, as it will not detect subclinical or mild hypothyroidism. Circulating T3 levels are normal in about 25% of patients, adaptive responses to hypothyroidism. T3 measurements are therefore not indicated.

Once clinical or subclinical hypothyroidism is confirmed, the etiology usually easily established by demonstrating the presence of TPO antibodies, which are present in 90-95% of patients with autoimmune hypothyroidism.

If there is any doubt about the cause of a goiter associated with hypothyroidism, FNAC can be used to confirm the presence of autoimmune thyroiditis.

ULTRA SONOGRAM

Because of the superficial location of the thyroid gland, high resolution real time gray scale and color Doppler sonography can demonstrate normal thvroid anatomy and pathologic conditions with remarkable clarity. As a result, this technique has come to play an increasingly important role in the diagnostic evaluation of thyroid diseases.

INSTRUMENTATION AND TECHNIQUE

High resolution (7.5-15.0 MHz) currently provide both deep ultrasound penetration up to 5cm- and high deformation images, with a resolution of 0.7 to 1.0 mm. No other imaging method can achieve this degree of spatial resolution.

The patient is typically examined in the supine position with the neck extended. A small pad may be placed under the shoulders to provide better exposure of the neck, particularly in patients with a short, stocky habitus. The thyroid gland must be examined thoroughly in both transverse and longitudinal planes. Imaging of the lower poles can be enhanced by asking the patient to swallow, which momentarily raises the thyroid gland in the neck. Regional lymph node enlargement also should be looked for.

FINDINGS:

In Hashimoto’ s thyroiditis, glandular enlargement is diffuse with a homogenous but coarsened parenchymal echotexture, generally more hypoechoic than a normal thyroid.

Fibrotic septations may produce a pseudolobulated appearance of parenchyma.

Multiple discrete, hypoechoic micronodules from 1 to 6 mm in diameter have been described as strongly suggestive of chronic thyroiditis. Histologically, they represent lobules of thyroid parenchyma which have been infiltrated by lymphocytes and plasma cells. These lobules are surrounded by echogenic fibrous strands. Micronodulation is a highly sensitive sign of chronic thyroiditis with a positive predictive value of 94.7%. But benign and ma1ignant thyroid nodules may coexist with chronic lymphocytic thyroiditis FNAC is often necessary to establish the final diagnosis.

Hypervascularity occurs when hypothyroidism develops.

Not infrequently, cervical lymphadenopathy is present especially affecting delphin node above thyroid isthmus.

The end-stage of chronic thyroiditis is atrophy where the gland is small with ill-defined margins, heterogenous texture due to progressive increase of fibrosis. Blood flow signals are absent. Occasionally discrete nodules occur and FNAC is needed to establish the diagnosis.

Although the appearance of diffuse parenchymal inhomogeneity and micronodularity is quite typical of Hashimoto's thyroiditis, other diffuse thyroid diseases most commonly adenomatous goiter, may have similar sonographic appearance. Some patients with adenomatous goiter have multiple discrete nodules separated by otherwise normal appearing thyroid parenchyma, other have enlargement with rounding of the poles of the gland, diffuse parenchyma, inhomogeneity and no recognizable normal tissue.

Ultrasound is also extremely useful to find out or rule out other pathologies particularly thyroid carcinoma. Ultrasound guided FNAC should be done whenever suspicion of malignancy arises.

FNAC

FNAC examination of the thyroid gland is occasionally useful in confirming the diagnosis of Hashimoto' s thyroiditis and in patients in whom malignancy is suspected.

The smears contain admixtures of lymphoid and epithelial cells in different proportions, depending on the stage of the disease.

Colloid is scant or absent.

In the late stage of the disease, fibrosis occurs and the aspirate may be poorly cellular.

The epithelial component shows a spectrum of nuclear and cytoplasmic changes from follicular cells with minimal oxyphilic alterations to fully developed Hurthle cells. The cytoplasm is denser than in normal follicular epithelial cells. The nuclei are enlarged and reveal variable degress of nuclear atypia. The degree of pleomorphism may be extreme. The follicular epithelium presumably as a result of TSH stimulation may also nuclear atypia and abundant cytoplasm. Without clinical and laboratory data, the cytologic features may be misinterpreted as indicative of a neoplastic process.

The lymphoid cells range from mature lymphocytes to small and large, cleaved and noncleaved, stimulated lymphocytes and immunoblasts.

Multinucleated giant cells are seen in 30% of cases.

When the lymphocyte component predominates, lymphoma should be considered. Lymphomas yield a monotonous, abnormal lymphoid population unlike the characteristic heterogenous lymphoid population of Hashimoto’s thyroiditis.

A lymphocytic infiltrate may co-exist with a neoplasm, particularly papillary carcinoma. Therefore special care should be taken in evaluating the follicular epithelial cells to exclude this possibility.

The role of FNAC in excluding a malignancy, when doubt arises cannot be overemphasized.

HISTOLOGY

Histologically, two variants of Hashimoto' s thyroiditis are recognized. - the classical and the fibrosing variety.

The classic type is characterized by a diffuse lymphocytic and plasmacytic cellular infiltrate followed by formation of well-developed germinal centers.

Thyroid follicles are small and can contain small amounts of colloid.

The epithelial cells are enlarged and contain abundant dense eosinophilic cytoplasm as well as a hyperchromatic large nucleus. These so- called Hurthle cell changes or metaplasia can be extensive, forming clusters or rests, and are frequently found near or mixed with lymphoid aggregates.

Fibrosis is apparent first in interlobular areas, but it can eventually replace areas of normal follicles.

The fibrosing variant account for about 10% of all cases of hashimoto's thyroiditis. Elderly patients present with large goitrous glands, severe hypothyroidism, and severe pressure symptoms. Grossly, the large well-

encapsulated glands have preservation of the lobular architecture. The histologic changes of typical Hashimoto's thyroiditis are intermingled with diffuse broad bands of fibrous tissue containing lymphocytes and plasma cells that extensively replace the normal parenchyma.

This variant can be distinguished from Riedel's thyroiditis because it does not extend beyond the thyroid capsule, the latter involves the thyroid as well as other neck structures.

A subacute variety of this condition causes transient pain and tenderness of the thyroid gland but in the majority of patients the thyroid enlargement changes over time from a rubbery consistency to stony hardness, associated with progressive hypothyroidism.

Hashimoto's thyroiditis can frequently be found coexisting with papillary carcinoma whether it represents an immunologic response to the tumor's antigens is unclear. However, most of the reported cases that favour this association lack serologic evidence of Hashimoto's thyroiditis. Therefore at present there is no evidence to support the malignant potential of this disease. Significant data do support the relationship between this process and the development of malignant lymphorna.

ESR

ESR is often raised in many patients with myxedema and 1-lashimoto's thyroiditis.

ISOTOPE SCAN

Thyroid isotope scanning usually reveals patchy uptake, and in general, provides little useful infonnation unless a dominant thyroid nodule is present. As with thyroid nodules in general, a dominant cold nodule in the setting of Hashimoto's thyroiditis should be evaluated with FNAC to rule out primary lymphoma of thyroid as there is an association between Hashimoto's thyroiditis and lymphoma of thyroid. Thus, lymphocytic subsets should be determined from the biopsy specimen if the more typical pathologic features of Hashimoto's thyroiditis are not present.

TREATMENT OF HASHIMOTO'S THYROIDITIS

There is no specific treatment for hashimoto's disease. Patients are usually followed medically, and replacement therapy is begun in patients with hypothyroidism or in patients with a goiter that is associated with pressure symptoms. Early initiation of thyroid hormone therapy is advised by many clinicians to prevent further thyroid enlargement and reduce the risk of myxedema.

Reduction in goiter size with thyroxine is more commonly seen in younger patients and in whom TSH is elevated.

For patients with obstructive symptoms, corticosteroids may be helpful.

Thyroxine therapy with long term follow-up monitoring of TSH levels is recommended.

Many clinicians believe that the circulatory level of TSH is the single most sensitive test of thyroid function.

If there is no residual thyroid function, the daily replacement dose of levothyroxine is usually 1.5 micrograms/kg body weight. In many patients, however, lower doses suffice until residual thyroid tissue is destroyed.

In elderly individuals who may have underlying myocardial ischemia, the initial dose should start at 12.5 or 25µgm/day, and the dose can be increased gradually.

The dose is adjusted on the basis of TSH levels, with the goal of treatment being a normal TSH, ideally in the lower half of their reference range. TSH responses are gradual and should be measured about two months after initiating treatment or after any subsequent change in levothyroxine dosage. The clinical effects of levothyroxine replacement are often slow to appear. Patients may not experience full relief from symptoms until 3 — 6 months after normal TSH levels are restored. Adjustment of levothyroxine is made in 12.5 — 25 micrograms increments if the TSR is high; decrements of the same magnitude should be made if the TSH is suppressed. Patients with a suppressed

TSH of any cause, including T4 overtreatment, have an increased incidence of atrial fibrillation and reduced bone density.

Once full replacement is achieved and TSH levels are stable, follow-up measurements of TSH is recommended at annual interval and may be extended to every 2 — 3 years, if a normal TSH is maintained over several years. It is important to ensure ongoing compliance, however, as patients do not feel any difference after missing a few doses of levothyroxine, sometimes leading to self-discontinuation.

It is not uncommon to see discordant T4-TSH values in patients being treated for primary hypothyroidism. This discordance results from the lag in the pituitary response to changing serum thyroid hormone levels. The serum TSR may take several weeks or months to return to normal despite normalization of serum T4 values with levothyroxine therapy. The physician must be aware of this response when assessing the thyroid status in patients being treated for primary hypothyroidism.

Patient education about the potential complications of mild, chronic hypothyroidism should help alleviate this worrisome practice.

INDICATIONS FOR SURGERY IN HASHIMOTO'S THYROIDITIS

Surgical intervention is indicated for

1. Obstructive symptoms,

2. Cosmetically unacceptable goiter,

3. When thyroid cancer (other than lymphoma) is found or suspected clinically.

DEQUERVAIN THYROIDITIS (SUBACUTE)

First reported in 1825, but de Quervain recorded its pathological description in 1904.4

De Quervain (subacute granulomatous) thyroiditis is the most common cause of a painful thyroid gland. It is a transient inflammation of the thyroid, the clinical course of which is highly variable. Most patients have pain in the region of the thyroid, which is usually diffusely tender, and some have systemic symptoms. Hyperthyroidism often occurs initially, sometimes followed by transient hypothyroidism. Complete recovery in weeks to months is characteristic.

Pathophysiology

A viral infection or a postviral inflammatory response is presumed to cause de Quervain thyroiditis. Serial studies of viral antibody titers have

implicated many viruses (including coxsackievirus, Ebstein-Barr mumps, measles, adenovirus, echovirus, and influenza), but the changes could be attributed equally to nonspecific anamnestic responses. Viral inclusion bodies are not observed in thyroid tissue.

A strong association exists with human leukocyte antigen (HLA)-B35 in most ethnic groups. A proposed mechanism is that the disease results from a viral infection that provides an antigen, either viral or resulting from virus-induced host tissue damage, that uniquely binds to HLA-B35 molecules on macrophages. The antigen-HLA-B35 complex activates cytotoxic T lymphocytes that damage thyroid follicular cells because they have some structural similarity with the infection-related antigen. The transient presence of autoantibodies (eg, inhibitory immunoglobulins that bind to thyrotropin [TSH], antibodies that block thyroid stimulation, thyroid antimicrosomal antibodies, thyroglobulin [TGB] antibodies) has been noted in the acute phase of the disease, but their presence is attributed to a virally induced autoimmune response and is not implicated in the pathological process. In contrast with autoimmune thyroid disease, the immune response is not self-perpetuating; therefore, the process is limited.

Destruction of follicular epithelium and loss of follicular integrity are the primary events in the pathophysiology of de Quervain thyroiditis. TGB, thyroid hormones, and other iodinatcd compounds are released into the blood, often in

quantities sufficient to elevate the serum thyroxine (T4) and triiodothyronine (T3) concentrations and suppress TSH secretion. This state lasts until the stores of TGB are exhausted or until healing occurs. New hormone synthesis temporarily ceases because of the low TSH. As inflammation subsides, the thyroid follicles regenerate and thyroid hormone synthesis and secretion resume. In some patients, several months are required for thyroid hormone synthesis to return to a normal rate; during that period, clinical hypothyroidism may be evident.

Clinical

History

Some patients experience a flulike prodromal episode 1-3 weeks prior to the onset of clinical disease. The natural course of the disease can be divided into the following 4 phases that usually unfold over a period of 3-6 months. The acute phase, lasting 3-6 weeks, presents primarily with pain. Symptoms of hyperthyroidism also may be present. The transient asymptomatic and euthyroid phase lasts 1-3 weeks. The hypothyroid phase lasts from weeks to months, and it may become permanent in 5-15% of patients. The recovery phase is characterized by normalization of thyroid structure and function.

The diagnosis is made based on clinical findings. Prodromal flulike symptoms or known infectious disease, such as pharyngitis, measles, mumps, Q

fever, or typhoid fever, may occur. In young patients, de Quervain thyroiditis may develop following an episode of Henoch-Schonlein purpura. However, a history of prodromal symptoms often cannot be obtained.

Local symptoms :- Pain over the thyroid area that is gradual or of sudden onset; that usually involves both lobes (in 30% of cases, it starts on one side and then migrates contralaterally within a few days); that radiates to the neck, ear, jaw, throat, or occiput; and is aggravated by swallowing and head movement; pain is the presenting symptom in over 90% of cases. Dysphagia, Hoarseness (uncommon)

Constitutional symptoms (often absent) :- Fever, Malaise, Anorexia, Fatigue, Muscle aches

Symptoms of hyperthyroidism:- (palpitations, tremulousness, heat intolerance, sweating, nervousness) occurring in the initial phase of the disease, Hyperthyroidism that usually is mild and rarely is severe, Transient symptoms, usually lasting 3-6 weeks

Symptoms of hypothyroidism:- occurring in the late phase of the disease in as many as half the cases, mostly mild or moderate, Transient hypothyroidism in 90-95% of cases

Hypothyroidism :- lasts weeks to months

Physical

Thyroid tenderness is usually symmetrical, but, occasionally, it starts in one lobe and then involves the contralateral lobe. Often, the pain is so severe that the patient cannot tolerate palpation of the neck. Thyroid enlargement is usually symmetric and mild, occasionally with areas of localized firmness.

Erytbema and hyperesthesia of the overlying skin may be present at the onset of severe cases. Cervical lymphadenopathy is uncommon. Hyperthyroidism.

Causes

A viral infection or a postviral inflammatory response in genetically predisposed individuals is presumed to cause de Quervain thyroiditis. Serial studies of viral antibody titers have implicated many viruses (including coxsackievirus, Ebstein-Barr, mumps, measles, adenovirus, echovirus, and influenza), but the changes could be attributed equally to nonspecific anamnestic responses. Recent studies failed to demonstrate significant chanves in serum antiviral antibody titers, or to detect viral DNA in the thyroid specimens.

INVESTIGATIONS

Laboratory Studies

Usually, the diagnosis is made on clinical grounds, and the only laboratory studies needed initially are those to determine whether hyperthyroidism is present, including TSH and free T4. If any doubt exists as to whether de Quervain thyroiditis is the correct diagnosis, 2 other tests may be helpful. Serum thyroglobulin is almost always markedly elevated. Erythrocyte sedimentation rate (ESR) is usually higher than 50mm/h in the initial phase. A normal or slightly elevated ESR makes the diagnosis of de Quervain thyroiditis relatively unlikely. ESR falls as the inflammatory process resolves, but following the ESR provides no useful information beyond what clinical observation yields.

After the initial inflammatory phase subsides, TSH should be monitored at intervals of 4-6 weeks for a few months to determine whether hypothyroidism occurs. Antibodies to TGB, thyroid peroxidase, and TSH receptor are usually absent in de Quervain thyroiditis and need not be sought unless the clinical differential diagnosis includes immune thyroid disease (Graves disease, chronic lymphocytic [Hashimoto] thyroiditis). In rare cases with systemic multiorgan involvement, elevation of serum alkaline phosphatase, gamma-glutamyl transpeptidase, aminotransferascs, and pancreatic enzymes may occur. Glucose intolerance has been reported.

Imaging Studies

- Ultrasonography of the thyroid gland

Ultrasonography provides no additional information and N rarely indicated for diagnostic purposes. In the first 3 phases of the disease, the thyroid gland is enlarged, shows unclear contour, and is diffusely or focally hypoechogenic. In the recovery phase, the thyroid structure and dimensions return to normal. Fibrosis is observed in some patients as hyperechogenicity and may occur as a form of healing. Extensive fibrosis is a predictor of hypothyroid state.

Doppler ultrasonography shows a near absence of vascularization in the acute phase and slightly increased vascularization in the recovery phase. During the acute phase, the more affected areas in the thyroid gland show the greatest decrease in vascularization, with the echogenically healthy-appearing regions of the thyroid showing normal or slightly increased vascularization. Ultrasonographic abnormalities are not correlated with the intensity of the inflammatory syndrome and/or thyroid function status. Recurrence can be seen as a new thyroid enlargement and an extension of hypoechoic regions. Risk of recurrence is not correlated with the initial ultrasonographic aspect, and there are no significant differences between patients with and without recurrence concerning the initial thyroid volume or echogenicity.

- CT scanning of the neck

CT scanning of the neck is not indicated for the diagnosis of thyroiditis. Consider that the administration of iodinated contrast material before measuring radioiodine uptake may result in a falsely decreased iodine uptake. If CT scanning is planned, it should be performed after any radioiodine uptake studies are completed.

The normal thyroid gland has a high attenuation (80-100 HU) because the normal thyroid gland concentrates iodine almost 100 times more than does the serum. In subacute thyroiditis, a diffusely swollen thyroid gland is observed, with a low attenuation corresponding to 45 HU. There is also moderate enhancement of the thyroid gland on contrast-enhanced scanning, suggesting the diffuse inflammatory nature of the disease process.

- **Magnetic resonance imaging:** MRI is not indicated for the diagnosis or evaluation of subacute granulomatous thyroiditis. If one is performed during the acute phase, the thyroid gland shows irregular margins and a higher than normal T1-weighted signal intensity and a much higher than normal T2-weighted signal intensity.

Neither radioiodine uptake (RAIU) nor thyroid scanning is indicated unless pain is mild or absent, in which case Graves disease might be considered in the differential diagnosis. RAIU is very low in the initial phase of subacute thyroiditis (<1-2% at 24 h) but usually is elevated in Graves disease.

Technetium-99m-pertechnetate scintigraphy typically demonstrates markedly reduced uptake in the thyroid gland during the acute stage, but this finding is not present in all patients. Technetium-99m-tetrafosmin uptake correlates with the stage of disease, particularly with inflammation, and shows increased uptake in the damaged area during the acute phase. However, this procedure is rarely used in the United States. Technetium-99m-sestamibi scanning may show diffuse increased uptake in the region of the thyroid gland, suggesting increased perfusion. The clearance rate of Technetium-99m-sestamibi during the early phase (ie, from 10 mm to 1 h) is decreased in the acute stage of subacute granulomatous thyroiditis

- **Fine-needle aspiration of the thyroid**

Fine-needle aspiration (FNA) is rarely needed; most of the time, the diagnosis of de Quervain thyroiditis can be made solely on clinical grounds. Some authors advocate that FNA should be performed in all patients with a tender thyroid to avoid misdiagnosis and inappropriate management. FNA is useful for diagnosis when atypical presentations of thyroid carcinoma and thyroid abscess are considered in the differential diagnoses. It usually shows the specific histological features of de Quervain thyroiditis (see Histologic Findings). FNA may provide unclear results in the acute stage when atypical follicular cells may appear in the aspirate, mimicking thyroid carcinoma.

Histologic Findings

Macroscopic

The thyroid gland is moderately enlarged and edematous in de Quervain thyruiditis. It may be unilaterally or bilaterally enlarged and has an intact capsule. Affected areas are firm find yellow-white and stand out from the more rubbery, normal, brown thyroid substance.

Microscopic

The changes are patchy and vary with the stage of the disease. The early phase is the active inflammatory phase and is characterized by areas of entirely disrupted follicles, which are replaced by neutrophils, forming microabscesses. In a later phase, the classic changes of granulomatous thyroiditis develop. This is characterized by aggregations of lymphocytes, large histiocytes, and plasma cells among damaged thyroid follicles. Multinucleated giant cells enclose pools or fragments of colloid, from which stems the designation giant cell thyroiditis. Colloid is also found within the giant cells, following a process called colloidophagy. In the final stages, the areas of injury are replaced by a chronic inflammatory infiltrate and fibrosis. Different histologic stages sometimes are found in the same gland, suggesting waves of destruction over a period of time.

Under a scanning electron microscope, the cytomorphology of subacute granulomatous thyroiditis shows loss of a uniform honeycomb cellular

arrangement, variation in size and decreased or shortened microvilli in follicular cells, and the appearance of round or ovoid giant cells. The giant cells are closely associated with the granulomas, and are $CD68^+$, thyroglobulin negative, and cytokeratin negative. The small lymphocytes in the granulomas are $CD3^+$, $CD8^+$, $CD45RO^+$ cytotoxic T cells. In the nongranulomatous lesions, the follicles are infiltrated by $CD8^+$ T lymphocytes, plasmacytoid monocytes, and histiocytes, resulting in disrupted basement membrane and rupture of the follicles. These findings suggest that cellular immune response may play an important role in the pathogenesis of subacute thyroiditis.

Treatment

Medical Management

Management is directed towards 2 problems-pain and thyroid dysfunction.

- Pain

Some patients with mild pain require no treatment. Nonsteroidal anti-inflammatory drugs (NSAIDs), such as ibuprofen (800-1200 mg/d in divided doses) or naproxen (1-1.5g/d in divided doses), or aspirin (2-4 g/d in divided doses) are used. Treatment can be tapered as allowed by the patient's pain. Analgesic therapy can usually be stopped after 2-6 weeks.

Steroids have no proven superiority to NSAIDs in de Quervain thyroiditis and should be avoided. If NSAIDs provide insufficient relief of pain within 3 days, then prednisone may be tried at 30-40 mg/d initially. The response to prednisone is usually prompt (within 2-3 d), and, if a prompt response does not occur, prednisone should be stopped. The dose of prednisone should be tapered slowly; symptoms often recur upon withdrawal.

If pain does not respond within 3 days, the diagnosis should be reconsidered.

- Management of thyroid dysfunction

In the initial phase of de Quervain thyroiditis, symptomatic hypcrthyroidiim can be treated with beta-blockade (propranolol 10-20mg qid or atenolol 25-0 mg/d), although mild symptoms may not require treatment. Beta-blockade, if needed, can usually be withdrawn in 2-6 weeks. Antithyroid drugs are of no value because the excess T4 and T3 results from release of preexisting thyroid hormone by follicular damage rather than from active synthesis.

If hypothyroidism occurs during the late phase, it is usually mild and transient. If symptoms are present or TSH is elevated, the patient needs replacement therapy with levothyroxine. Depending on the level of TSH, the starting dose can be 25-100 mcg/d and is adjusted for normalization of TSH. Usually, the hypothyroid stage lasts 2-3 months, but some authors recommend treatment for as long as 6 months, followed by discontinuation and monitoring

of TSH. Rare cases with permanent hypothyroidism require lifelong replacement therapy.

Surgical Management

No surgical treatment is required in de Quervain thyroiditis.

Diet

Diet modification has no role in the management of de Quervain thyroiditis.

Medication

The goals of pharmacotherapy are to reduce disease-associated morbidity and prevent complications due to abnormal thyroid function. No treatment has been shown to impact the occurrence of permanent hypothyroidism.

Salicylates

Used for symptomatic treatment. Aid in the relief of mild to moderate pain by inhibiting inflammatory reactions and pain.

Aspirin (Anacin, Ascriptin, Bayer Aspirin, Bayer Buffered)

NonsteroidaI anti-inflammatory drugs

Although most NSAIDs are used primarily for their anti-inflammatory effects, they are effective analgesics and are useful for the relief of mild to moderate pain.

Naproxen(Aleve, Naprelan, Naprosyn, AnaProx)

For relie\of mild to moderate pain. Inhibits inflammatory reactions and pain by decreasing activity of\cyclooxygenase, which results in a decrease of prostaglandin synthesis.

Glucocorticoids

Have anti-inflammatory properties and cuase profound and varied metabolic effects. They modify the body's immune response to diverse stimuli.

Beta-adrenergic blocking agents

Used for symptomatic relief of hyperthyroidism. Propranolol is used as an example, but any noncardioselective beta-blocker can be used. Use only in moderate to severe cases of hyperthyroidism, and many times it is not needed.

Propranolol (Inderal, Betachron E-R)

Thyroid hormones

Required during the recovery phase if transient hypothyroidism occurs. Required indefinitely in the occasional patient who develops permanent hypothyroidism.

Levothyroxine (Levothroid, Levoxyl, Synthroid)

DOC. In active form, influences growth and maturation of tissues. Involved in normal growth, metabolism, and development.

Adult

Transient hypothyroidism (symptom relief): 25-100 mcg/d PO

Permanent hypothyroidism: 1.6 mcg/kg PO

Pediatric

Neonate to 6 months: 25-50 mcg/d PO

6-12 months: 50-75 mcg/d PO

1-5 years: 75-100 mcg/d PO

6-12 years: 100-150 mcg/d PO

>12 years: 150 mcg/d PO

Follow-up

Inpatient care is only recommended in the rare cases in which severe symptomatic hyperthyroidism is present. The authors recommend monitoring thyroid function every 2-4 months for 1 year or until thyroid function normalizes (whichever occurs later). Yearly monitoring of thyroid function should be performed thereafter for several years, since permanent hypothyroidism may occur several years following the initial diagnosis.

Complications

- **Acute complications**

Severe hyperthyroidism may be observed during the inflammatory phase (see History). Multiple system organ failure may complicate the course of the

disease in exceptionally rare cases. Pancreatitis or splenomegaly has been associated with de Qucrvain thyroiditis in case reports only. Vocal cord paralysis occurs occasionally in cases with severe thyroid gland inflammation. Cerebral venous thrombosis was reported in some cases. In one case, the patient was a heterozygous carrier for the G202 10A mutation of the prothrombin gene, which predisposed her to this complication.

- **Long-term complications**

Permanent hypothyroidism is the most frequent long-term complication of de Quervain thyroiditis. It is observed in less than 510% of the patients and requires thyroid replacement therapy. Disease recurrence has been documented in occasional cases (up to 20% of cases in some series). Recurrence is more frequent in the first year but has been reported even 30 years after the initial diagnosis. The risk of recurrence cannot be correlated with initial thyroid function, inflammatory syndrome, or ultrasonographic aspect (ie, thyroid volume. echogenicity).

Prognosis

Prognosis is excellent. More than 90% of the patients with de Quervain thyroiditis recover completely, with or without treatment. Even in the 5-10% of patients who develop hypothyroidism, treatment with thyroid hormone is effective.

RIEDEL'S THYROIDITIS

Riedel thyroiditis, or Riedel's thyroiditis (RT), is a rare, chronic inflammatory disease of the thyroid gland characterized by a dense fibrosis that replaces normal thyroid parenchyrna. The fibrotic process invades adjacent structures of the neck and extends beyond the thyroid capsule. This feature differentiates RI from other inflammatory or fibrotic disorders of the thyroid. Because of the encroachment beyond the thyroid capsule, other problems can be associated with RI, including hypoparathyroidism, hoarseness (due to recurrent laryngeal involvement), and strider (due to tracheal compression). Some experts feel that RT is not primarily a thyroid disease but rather that it is a manifestation of the systemic disorder rnultifocal fibrosclerosis. Approximately one third of RT cases are associated with clinical findings of multifocal fibrosclerosis at the time of diagnosis.

In 1883, Professor Bernhard Riedel first recognized the disease. He published a description of 2 cases in 1896 and of a third case in 1897.1 Riedel used the term eisenharte strums to describe the stone-hard consistency of the thyroid gland and its fixation to adjacent structures. He noted the presence of chronic inflammation with fibrosis and the absence of malignancy on microscopic examination. Simple wedge resection of the thyroid isthmus was

used to alleviate tracheal obstruction and is still the preferred surgical therapy for RT.

Pathophysiology

The etiology of Rieders thyroiditis (RT) is unknown. One theory of pathogenesis postulates that RT results from an autoimmune process. A second theory holds RT to be a primary fibrotic disorder.

The following evidence supports an autoimmune pathogenesis for RT:

The presence of antithyroid antibodies in a significant percentage of patients with RT (67% of 178 cases reviewed in one study). The pathological features of cellular infiltration, including lymphocytes, plasma cells, and histiocytes. The frequent presence of focal vasculitis on pathologic examination. The favorable response of a subset of patients with RI to treatment with systemic corticosteroids

However, the presence of normal lymphocyte subpopulations and normal serum complement levels weighs against an autoimmune mechanism. Additionally, elevated levels of antithyroid antibodies may merely reflect the immune system’s exposure to sequestered antigens released by the destruction of thyroid parenchyma from a primary fibrotic disorder.

The theory that RT is a primary fibrotic disorder is supported by its association with rnultifocal fibrosclerosis. This uncommon idiopathic syndrome

is characterized by fibrosis involving multiple organ systems. The extra cervical manifestations of multifocal fibro sclerosis can include retroperitoneal fibrosis, mediastinal fibrosis, orbital pseudotumor, pulmonary fibrosis, sclerosing cholsngitis, lacrimal gland fibrosis, and fibrous parotitis. RI may be but 1 manifestation of this multifocal disease.

The histopathologic changes of RT closely resemble those observed in multifocal fibrosclerosis. Additionally, one third of published RI cases have demonstrated at least one manifestation of extracervical fibrosclerosis. The ability of systemic corticosteroids and tamoxifen to inhibit fibrogenesis accounts for the favorable effect of such treatment in both conditions.

CLINICAL

History

Riedel's thyroiditis (RT) is characterized by the replacement of normal thyroid parenchyma with dense fibrotic tissue and by the extension of this fibrosis to adjacent structures of the neck. Patients typically present with a hard, fixed, painless goiter. The character of the thyroid gland is often described as stony or woody. The onset of the goiter may be sudden, but it is usually gradual. Involvement may be unilateral or bilobar. Thyroid function depends on the extent to which the normal thyroid gland has been replaced by fibrotic tissue. Most patients are euthyroid. Hypothyroidism is noted in approximately 30% of

cases. Rarely, hyperthyroidism can occur, but this is probably secondary to a coexisting condition.

Local compressive symptoms, such as neck tightness or pressure, dyspnea, dysphagia, hoarseness, choking, and cough, are frequent. Such symptoms are the result of the increasing thyroid mass or are due to the extension of the fibrotic process to adjacent neck structures (eg. strap muscles, trachea, esophagus, recurrent laryngeal nerve). Hypoparathyroidism is rare and presumably reflects fibrotic involvement of the parathyroid glands. Recurrent laryngeal nerve paralysis is also uncommon, but it can be observed in extensive disease. Occasionally, spontaneous remission has been reported. Patients can also relapse.

Physical

Clinical features of Riedel's thyroiditis (RT) closely resemble those of anaplastic carcinoma of the thyroid. Patients note a nonpainful, rapidly growing thyroid mass. One distinguishing feature of RT is the absence of associated cervical adenopathy. However, accurate diagnosis requires open biopsy. RT and anaplastic carcinoma of the thyroid can be distinguished by mmunohistochemistry.

Approximately one third of patients with RT have an associated extracervical manifestation of multifocal fibroscierosis (eg. retroperitoneal

fibrosis, mediastinal fibrosis, orbital pseudotumor, pulmonary fibrosis, scierosing cholangitis, lacrimal gland fibrosis, fibrosing parotitis).

Causes

The etiology of Riedel's thyroiditis is unknown.

INVESTIGATIONS

Laboratory Studies

The laboratory findings of Riedel's thyroiditis (RT) are nonspecific. The erythrocyte sedimentation rate (ESR) is generally elevated. Most patients remain euthyroid. Approximately 30% of patients become hypothyroid. Rarely, patients are hyperthyroid. In one review, antithyroid antibody levels (TG-Ab and TPO-Ab) were found to be elevated in 67% of 170 cases.2 However, it is not certain whether such autoantibodies are pathogenic or whether their presence merely reflects exposure of the immune system to sequestered antigens released by the fibrotic destruction of normal thyroid parenchyma.

Imaging Studies

In cases of Riedel's thyroiditis (RT), imaging studies may suggest the diagnosis, but findings can be nonspecific. Enlargement of the affected thyroid gland and compression or invasion of adjacent structures, such as the strap muscles, trachea, esophagus, or carotids, may be observed on computed

tomography (CT) or magnetic resonance imaging (MRI) scans. However, these studies cannot reliably distinguish between RT and invasive thyroid malignancy.

CT scanning shows affected areas of the thyroid to be hypodense. The area is usually isodense with the neck muscles. The use of iodinated contrast has occasionally been reported as causing increased enhancement, but usually it is decreased, especially if extensive fibrosis is present. On MRI scans, the affected thyroid gland is typically hypointense on T1- and T2-weighted images. Decreased enhancement has usually been reported with gadolinium contrast use, but occasionally.

Medical Management

The rarity of Riedel's thyroiditis (RT) makes controlled studies of RT therapy impractical. Recommendations for medical treatment have been largely based on empirical experience.

Currently, corticosteroid therapy is the medical treatment of choice for patients with RT. Most studies note a reduction in goiter size and the relief of local compressive symptoms, although some patients show no benefit.

Some investigators believe that a favorable response is more likely early in the course of the disease. Improvement is less likely to occur in patients with

advanced RT when the affected portions of the thyroid gland have been completely replaced by fibrotic tissue.

Corticosteroids are believed to act by reducing inflammation and by inhibiting the actions of fibrinogenic cytokines. No consensus has been reached on the corticosteroid dosing regimen. However, all studies advocate an initially high dose to alleviate compressive symptoms, followed by gradual tapering over months to a lower maintenance dose.

The effectiveness of therapy can be judged by symptomatic improvement and by following the reduction of the ESR and the thyroid autoantibody levels. Many patients can be weaned from therapy, but others require more prolonged treatment.

Tamoxifen has been used in RT patients as a first-line therapy, but it has also been employed after the failure of corticosteroid treatment. The usual dose that has been found to be effective is 20 mg taken orally twice a day. Patients who respond can be tapered to 20 mg once each day or to 10 mg twice a clay. Because of the relatively infrequent occurrence of RT, comparison studies with tamoxilen and steroid therapy have not been undertaken. Few and colleagues initially advocated tamoxifen in a study of 4 patients with progressive RT who were not responsive to corticosteroids or surgical decompression Each patient had a decrease of 50% or more in the size of the thyroid mass, with one patient having total resolution. Since then, additional reports have described successful

treatment of EU with tamoxifen. An oral dose of 20 my twice a day provided each patient with symptomatic improvement, as well as a reduction in the size of the involved tissue as measured on CT scan.

Estrogen receptors have not been demonstrated in RT tissue. Therefore, the mechanism of action was not proposed to be tamoxifen's antiestrogen activity but rather its induction of transforming growth factor beta, a potent inhibitor of fibroblast proliferation. Use levothyroxine therapy to correct hypothyroidism associated with RT. Many authorities advocate not only thyroid replacement but also suppression of thyroid-stimulating hormone (TSH) in all patients with RT, regardless of thyroid function. However, the degree to which TSI-I stimulates the fibroinflammatory processes of RT, if at all, is unknown. As such, recommendations for TSH suppression must be regarded as empirical.

Surgical Management

Surgery for patients with Riedel's thyroiditis (RT) serves the dual purpose of establishing the diagnosis and relieving tracheal compression. A wedge resection of the thyroid isthmus remains the preferred method for accomplishing these ends. Surgery is indicated when tissue is needed for diagnosis, medical treatment shows no benefit, or compressive symptoms are very severe.

Open surgical biopsy is essential to the definitive establishment of the diagnosis of RT and to the exclusion of carcinoma.

A wedge resection of the isthmus relieves tracheal compression. Grossly, the affected tissue is stony and hard and is white or pale gray. It has a similar feel to cartilage when incised.

More extensive thyroid surgery is generally discouraged because extra thyroid fibrosclerosis alters the anatomy and obliterates surgical planes. The trachea, esophagus, carotids, recurrent laryngeal nerves, or parathyroid glands may be encased by fibrotic tissue and are at increased risk for iatrogenic surgical damage.

Medication

The goals of pharmacotherapy are to reduce morbidity and prevent complications.

Corticosteroids

Have anti-inflammatory properties and cause profound and varied metabolic effects. Corticosteroids modify the body's immune response to diverse stimuli.

Prednisone (Deltasone, Meticorten, Orasone, Sterapred)

Immunosuppressant for the treatment of autoimmune disorders. Prednisone may decrease inflammation by reversing increased capillary

permeability and suppressing PMN activity. The drug stabilizes lysosomal membranes and suppresses lymphocytes and antibody production.

Antineoplastic agents

For progressive Riedel's thyroiditis that is not responsive to corticosteroids or surgical decompression, on for with contraindications to corticosteroid therapy. Antineoplastic agents provide symptomatic improvement, well as size reduction of the involved tissue as measured on CT scans.

Thyroid hormones

Used to correct hypothyroidism associated with RiedeEs thyroiclitis.

Levothyroxine (Levothroid, Levoxyl, Synthroid, Unithroid)

Rapidly inhibits the release of thyroid hormones via a direct effect on the thyroid gland; it also inhibits the synthesis of thyroid hormones. Iodide also appears to attenuate cAMP-mediated effects of TSH. In active form, levothyroxine influences the growth and maturation of tissues. It is involved in normal growth, metabolism, and development.

Follow up

The patient with Riedels thyroiditis should be followed for progression of the disease and for the development of multifocal fibrosclerosis. Repeat imaging of the neck by CT scanning or MRI should be performed at intervals defined by the rate of progression.

The patient's TSH level should be routinely checked and maintained in the reference range, with levothyroxine administered as necessary.

Complications

- Airway obstruction
- Dysphonia
- Hoarseness
- Hypothyroidism
- Hypoparathyroidism
- Dysphagia

Prognosis

Riedel's thyroiditis (RT) is generally a self-limited disease with a favorable prognosis. Death due to airway compromise is very rare. One third of patients with RT ultimately develop at least 1 extracervical manifestation of multifocal fibrosclerosis (such as retroperitoneal fibrosis. mediastinal fibrosis, or sclerosing cholangitis. In such patients, the prognosis essentially becomes that of extra cervical fibrosclerosis. Therefore, when RT is diagnosed, it is essential to perform abdominal and chest imaging studies to exclude concomitant, extra cervical entities from multifocal fibro sclerosis.

ACUTE (SUPPURATIVE) THYROIDITIS

The thyroid gland is inherently resistant to infection as a consequence of its extensive blood and lymphatic supply, high iodide content, and fibrous capsule, but infectious agents can seed it (1) via the hematogenous or lymphatic route; (2) via direct spread from persistent pyriform sinus fistulae or thyroglossal duct cysts; (3) as a result of penetrating trauma to the thyroid gland: or (4) as a result of immunosuppression. Streptococcus and anaerobes account for about 70% of cases, but escherichia coli, Pseudomonas aeruginosa, Haemophilus influenzae. Eikenella Corrodens, corynebacterium, and Coccidimyosis species also have been cultured. Acute suppurative thyroiditis is more common in children and is often preceded by an upper respiratory tract infection or otitis media. It is characterized by severe neck pain radiating to the jaws or ear lever, chills, odynophagia and dysphonia. Rarely, complications such us systemic sepsis, tracheal or esophageal rupture, jugular vein thrombosis, laryngeal chondritis or perichondritis and sympathetic trunk paralysis may result.

The diagnosis is established by leukocytosis on blood tests and FNA biopsy for Gram's stain, culture, anti cytology. CT scans may help to delineate the extent of infection. A persistent pyriform sinus fistula should always be

suspected in children with recurrent acute thyroiditis. A barium swallow demonstrates the anomalous tract with 80% sensitivity.

Treatment consists of parenteral antibiotics and drainage of abscesses. Patients with pyriform sinus fistulae require complete resection of the sinus tract, including the area of the thyroid where the tract terminates, in order to prevent recurrence.

DRUG INDUCED THYROIDITIS

Many medication can alter thyroid function or the results of thyroid function tests. However, only few, are known to provoke autoimmune or destructive inflammatory thyroiditis.

AMIODARONE

Amodarone induced hypothyroidism occurs in patients with pre existing thyroid autoimmunity, while receiving amiodarone, Treatment is given with levothyroxine sodium and amiodarone may be continued.

Amiodarone induced thyrotoxicosis occurs in upto 23% of patients receiving amiodarone.

LITHIUM

In patients with pre existing thyroid autoimmunity lithium may increase serum thyroid antibody concentrations and lead to subclinical or overt hypothyroidism. The prevalence ranges from 10-33%.

RADIATION THYROIDITIS

It is caused either during treatment with radio active iodine for hyperthyroidism or while receiving external beam radiation it is for certain cancers. It is therapy more frequently, manifests as hypothyroidism and occasionally thyrotoxicosis. Diagnosis is made by altered thyroid function Levothyroxine sodium is given as treatment.

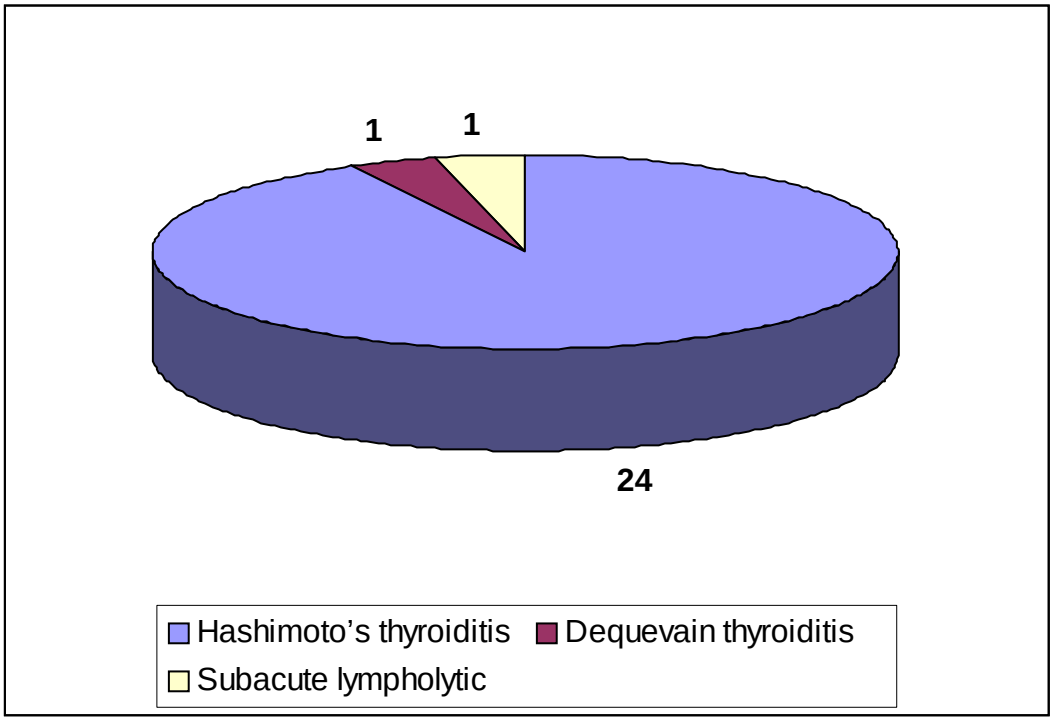
POST-PARTUM THYROIDITIS

These disorders are similar and follow the same general clinical course of thyrotoxicosis to owed by hypothyroidism. The only real difference between them is that post-partum thyroiditis occurs after the delivery of a baby while painless thyroiditis occurs in men and in women not related to a pregnancy Not all patients demonstrate evidence of going through both phases; approximately 1/3 of patients will manifest both phases: while 1/3 of patients will have only a thyrotoxic or hypothyroid phase. The thyrotoxic phase lasts for 1-3 months and is: associated with symptoms including anxiety, insomnia, palpitations (fast heart rate) fatigue, weight loss, and irritability The hypothyroid phase typically occurs 1-3 months after the thyrotoxic phase and nay last up to 9-12 months. Typical symptoms include fatigue, weight gain, constipation, dry skin, depression and poor exercise tolerance. Must patients (~80%) will have return of their thyroid function to normal within 12-18 months of the onset of symptoms.

PRESENTATION OF THE ANALYTICAL STUDY

Table 1
Incidence of type of thyroiditis in goitrous patients

Types	Incidence
Hashimoto's thyroiditis	24
Dequerain thyroiditis	1
Subacute lymphocytic	1

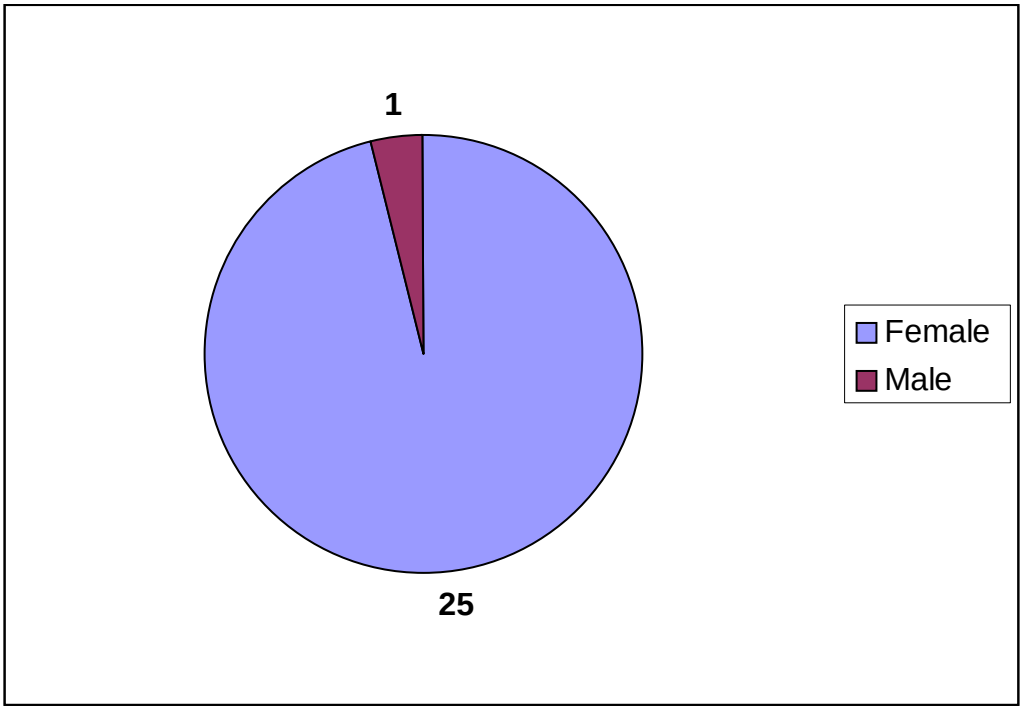


This analysis shows that most common type of thyroiditis is hashimotos thyroiditis.

Table 2
Sex distribution

Sex	No. of patients
Female	25
Male	1
Total	26

Female 10 male ratio was observed as 25 :1



Graph showing sex distribution sex distribution of thyroiditis

Table 3
Age distribution

Age (in yrs)	Female	Male
< 20	1	
20-29	4	
30-39	16	
40-49	4	1
> 50	1	

The age of patient average from 18-55yr this analysis shows that leak incidence of thyroiditis is 30-40 years.

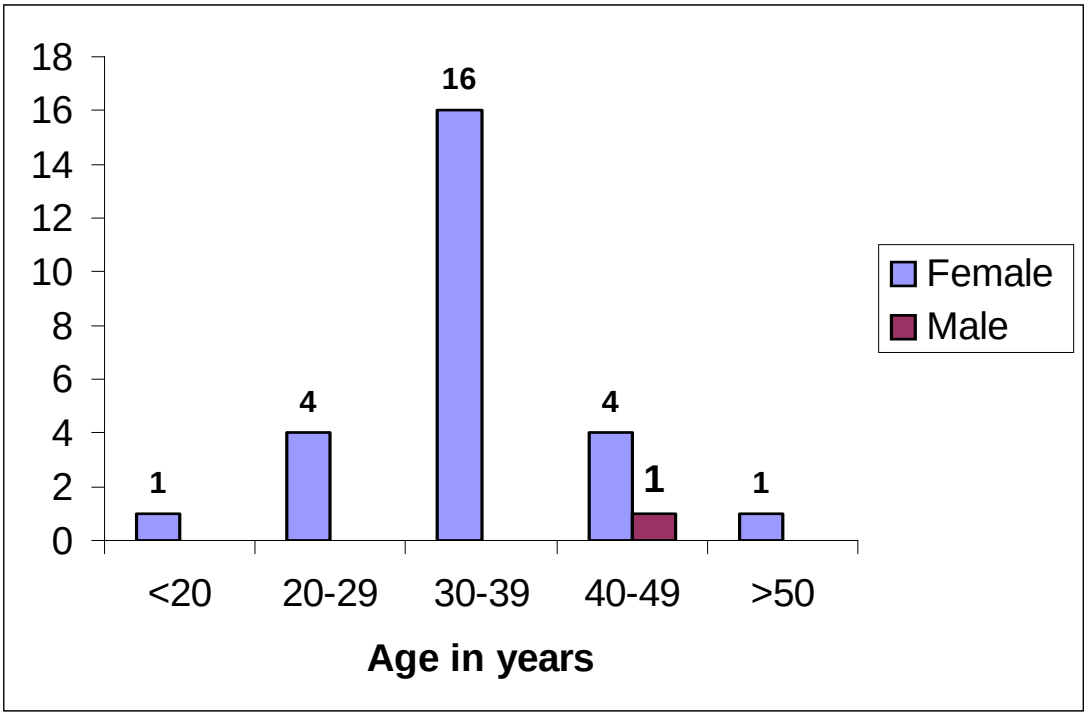


Table 4
Mode of presentation

Symptoms	**No. of patients**
Swelling	26
Compressive symptom	4
Pain	2
Change in voice	-
Toxic	-

The analysis shows that all 26 patient percent with history of swelling in front of neck. 2 patients with history of pain associated with swelling.

Table 5
Clinical presentation

Clinical presentation	No. of patients
MNG	20
Diffuse enlargement	4
Solitary nodule	2

Out of 26 cases, 20 cases presented with MNG 4 cases as diffuse enlargement, 2 cases presented as solitary nodule.

Graph showing clinical presentation

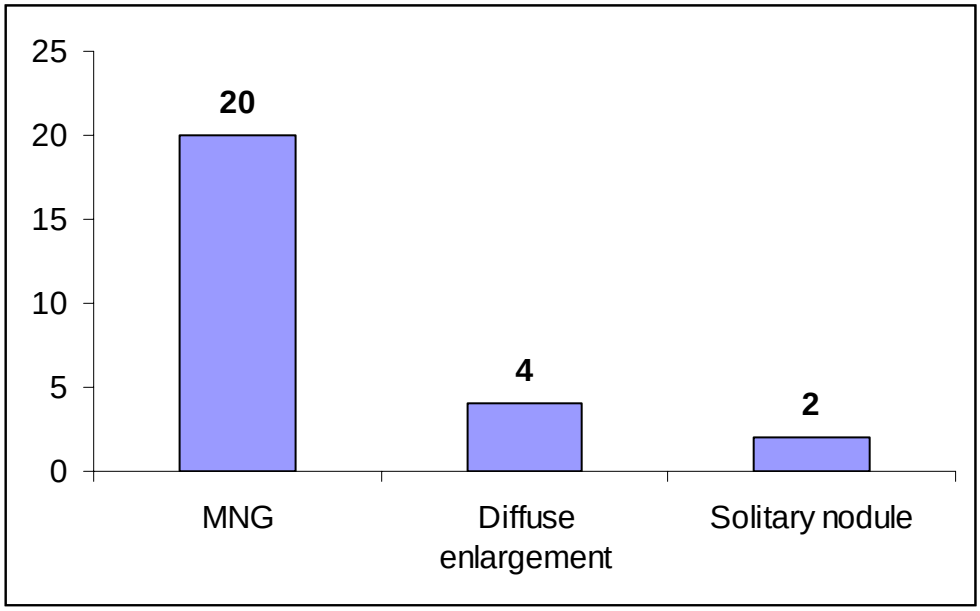


Table 6
Hormonic status

Thyroid status	No. of patients	Percentage
Hypothyroid	9	34.6
Euthyroid	16	61.5
Hyperthyroid	1	3.8

Out of 26 cases. 16 cases had erythroid, 9 cases are hypothroid a 1 case had hyperthyroid.

Graph shows thyroid hormonal status

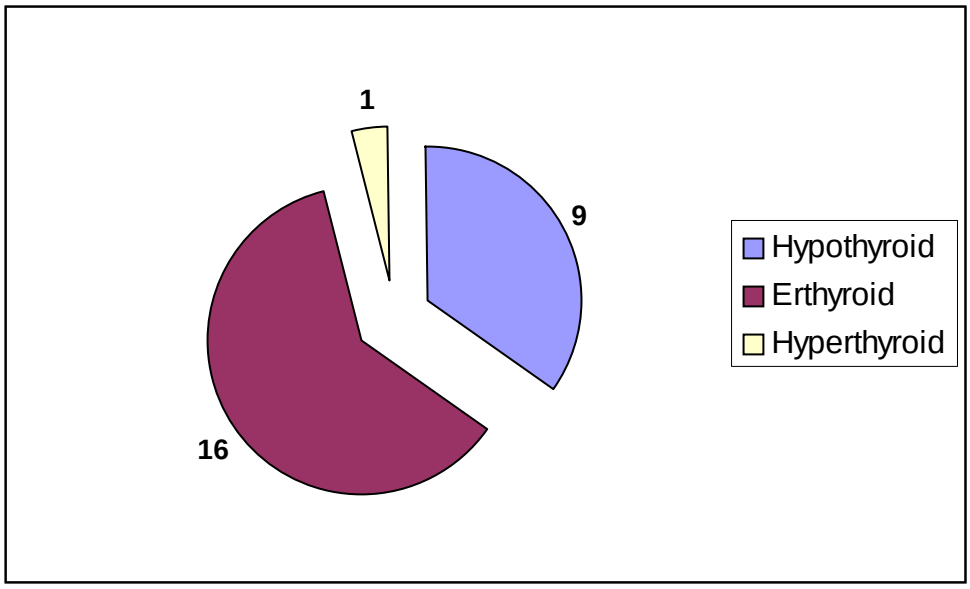


Table 7

FNAC status at presentation

FNAC	No. of patients
Benign follicular cells	4
Hashimoto's thyroditis	20
Haemorrhagic smear	2

Out of 26 patient, 20 patients of cytopathologial diagnosis of thyroidits as hashimoto's thyroiditis.

Table 8

Antimicrosomal Antibodies

In this study a positive antimicrosomal antibodies was positive for only 20 patients.

Table 9

ESR status

	No. of patients	**Percentage**
Raised	14	53.8
Normal	12	46
Total	26	100

ESR was found to be elevated in about 53.8% of the cases.

Table 10

Postoperative diagnosis

	No. of patients
Preop diagnosis	22
Post op diagnosis	4

Out of 26 cases, three cases FNAC found as colloid goitre, for subtotal thyroidectomy done and histopathology report was hashimotos thyroidits another one case was operated for swelling which did not reduce in size. 22 cases are treated medically.

DISCUSSION

During the study period from 2007-2009 out of 236 goitrous patients, 26 patient were diagnosed as thyroidits. Hashimoto's thyroiditis is the most common type of thyroiditis.

Out of 26 patients 24 patients was diagnosed as Hashimotos. One patient was diagnosed as dequervain thyroiditis and one cases was diagnosed as lymphocytic thyroditis.

Female preponderance is a well established feature of thyroid, our study have female preponderance, male to female ratio being 1:25.

In our study age incidence range from 18-55 yrs. The highest incidence being in 30-39 yrs.

All patients presented with a history of swelling in front of neck two patients were presented with dysphagia, another two patients were presented with pain over the thyroid region.

In our study, 20 cases are presented as multinodular swelling, 11 patients were presented with diffuse enlargement and 2 cases were presented as solitary nodular swelling.

In our study 16 patients are presented as euthyroid, 9 patients were hypothyroid and one patient with hyperthyroid. Out of 26 cases, 20 cases were diagnosed by FNAC as Hashimoto's Thyrodiits and two patients as haemorrhagic smear.

In our study about 20 of the cases had thyroid autoantibiodies positive and ESR positivity about 53.8%.

The patients were followed up regularly at interval of 3 months about for 2 years, at every visit pulse, weight, circumference of neck were recorded.

All hypothyroid and euthyroid patient were put on ELTROXIN replacement (dose- 100µg) after treatment all patients were found to clinically euythroid after 3 months.

All patients with diffuse goitre and solitary nodule put on throxine showed a decrease in size of gland that was appreciated by measuring the girth of neck.

Four patients underwent surgery, of these three were colloid goitre and under went subtotal thyroidectomy, later diagnosed as hashimoto's thyroidits by post operative histopathology. Surgery done for one patient for compressive symptoms.

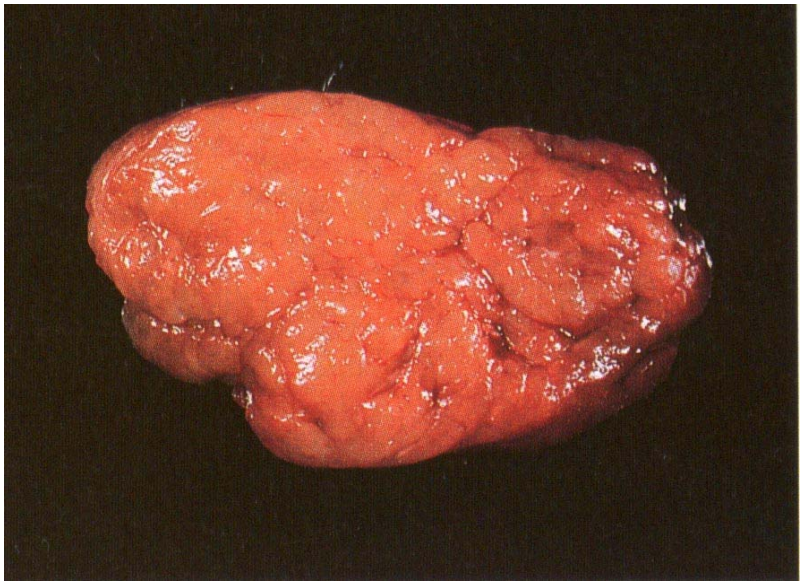
CONCLUSION

- Hashimoto’s thyroiditis is a most common type of thyroidits in goitrous patient.
- Female are more prone to develop
- Maximum incidence age group of about 30 to 40 years.
- Thyroiditis has a varied presentation as diffuse goitre, multinodular goitre or a Solitary nodular goitre.
- Thyroiditis could present as in hypothyroid, euthyroid and hyperthyroid state.
- Diagnosis of thyroiditis could be done by FNAC, Antibody titre, ESR, histopathology report.
- Treatment is primarily medical with thyroxine replacement and surgery is required with patient having pain, difficulty in swallowing and not regressing with eltroxin.

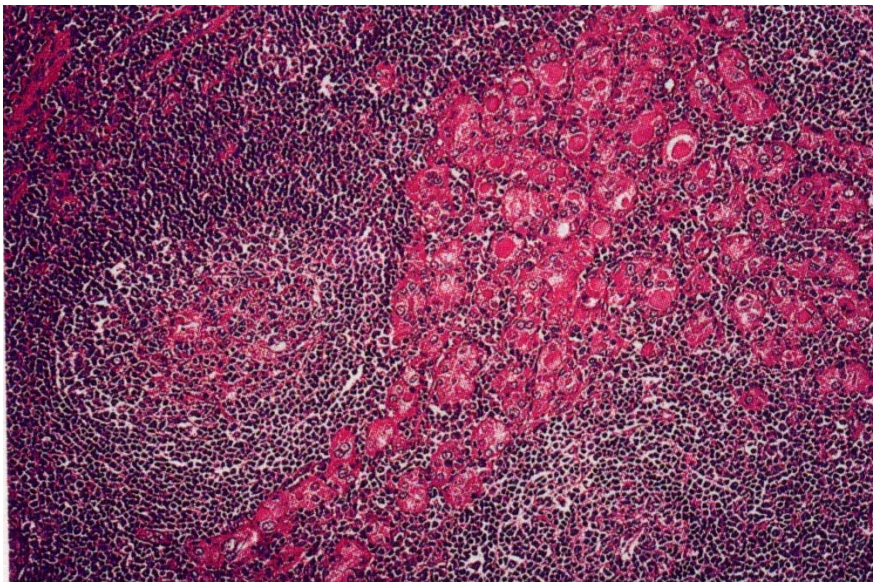
Characteristics	Hashimotos	Painless postpartum	Subacute	Suppurative	Riedel's
Age at onset (yrs)	All ages	Childbearing age	20-60	Children 20-40	30-60
Sex ration (F:M)	8-9:1	-	5:1	1:1	3-4:1
Cause	Auto immune	Auto immune	Unknown	Infectious	Unknown
Pathological findings	Lymphocytic infiltration, germinal centers	Thyrotoxicosis, hypothyroid or both	Thyrotoxicosis, hypothyroid Or both	Usually euthyroidism	Usually euthyroidism
TPO	High titer	High titer	Low titer	Absent	Usually

TYPE	CAUSE	CLINICAL FEATURES	DIAGNOSIS (Not All Tests May Be Needed)	DURATION AND RESOLUTION
Hashimoto's thyroidits	Anti-thyroid antibodies, autoimmune disease	Hypothyroidism, rare cases of transient tinyrotoxicosis	Thyroid function tests, thyroid, anti body test	Hypothyroidism is usually permanent
Subacute thyroditis (de Quervain's thyroiditis)	Possible viral cause	Painful thyroid, thyrotoxicosis followed by hypothyroidism	Thyroid function tests, sedimentation rate, radio active iodine uptake	Resolves to normal thyroid function within 12-18 months, 5% possibility of permanent hypothyroidism.
Silent thyroiditis, [Painless thyroditis]	Anti thyroid antibodies, autoimmune disease	Thyrotoxicosis followed by hypothyroidism	Thyroid function tests, thyroid antibody tests, radio active iodine uptake	Resolves to normal thyroid function within 12-18 months, 20% possibility of permanent hypothyroidism.
Post partum thyroiditis	Anti thyroid antibodies, autoimmune disease	Thyrotoxicosis followed by hypothyroidism	Thyroid function tests, thyroid antibody tests, radio active iodine	Resolves to normal thyroid function within 12-18 months, 20%

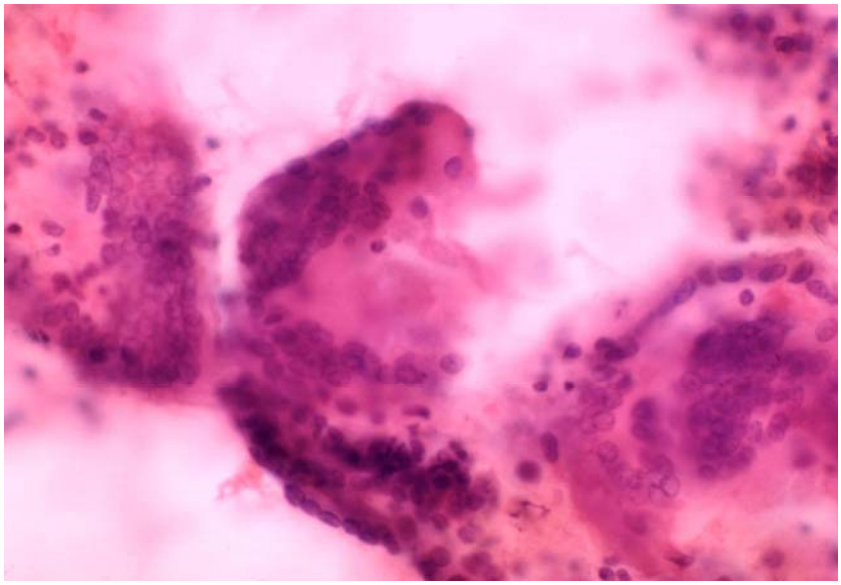
			uptake(contra indicated if the women is breast – feeding)	possibility of permanent hypothyroidism.
Drug induced	Drug include amiodarone, lithium, interferons, cytokines	Either thyrotoxicosis or hypothyroidism	Thyroid function tests, thyroide antibody tests	Often continues as long as the drug is taken
Radiation induced	Follows treatment with radioactive iodine for hyperthyroidism or external beam radiation therapy for certain cancers.	Occasionally thyrotoxicosis, more frequently hypothyroidism	Thyroid function tests	Thyrotoxicosis is transient, hypothyroidism is usually permanent
Acute thyroiditis, suppurative thyroiditis	Bacteria mainly but any infectious organism	Occasionally painful thyroid, generalized illness, occasional mild hypothyroidism	Thyroid function tests, radioactive iodine uptake, fine needle aspiration, biopsy	Resolves after treatment of infectious cause, may cause severe illness



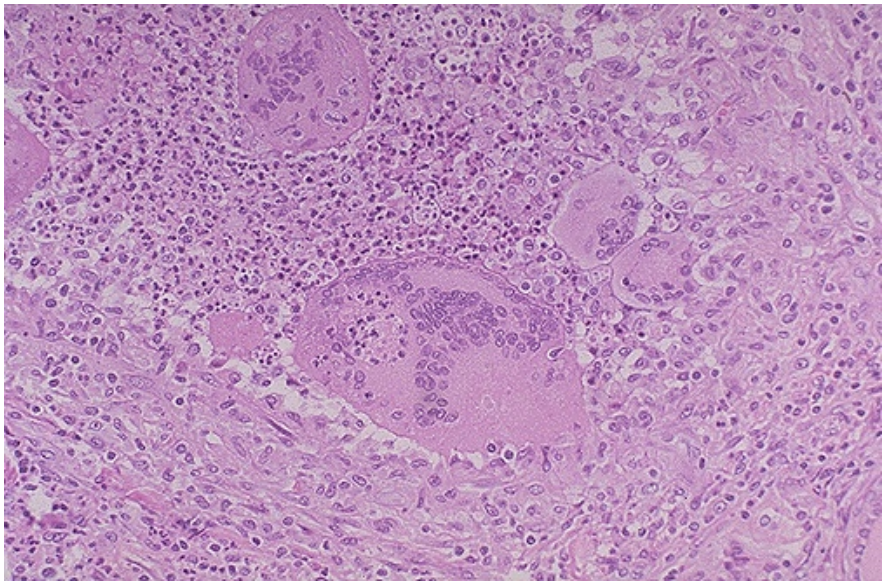
RESECTED SPECIMEN OF THYROID



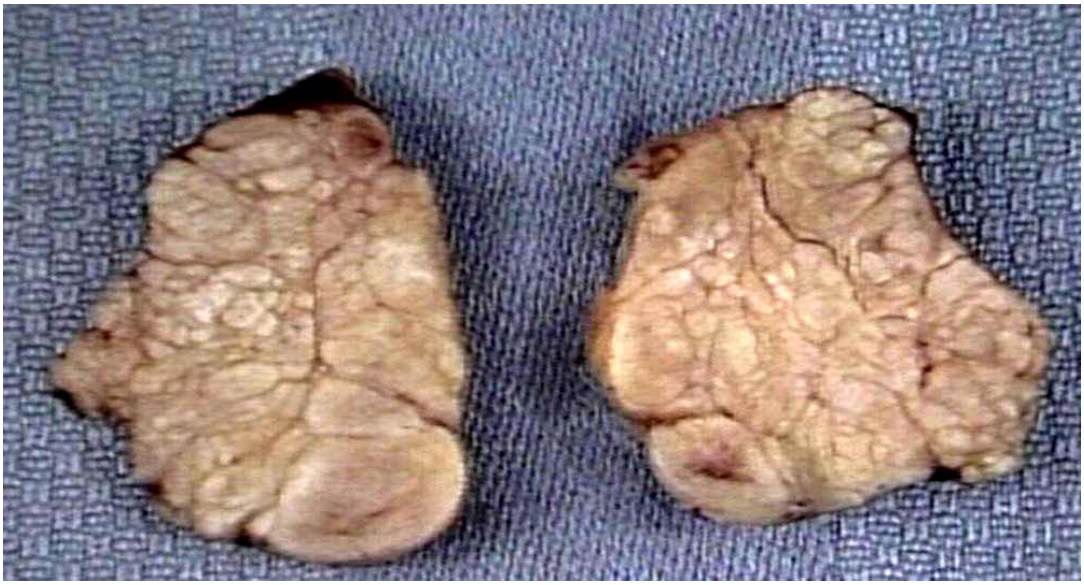
HISTOPATHOLOGY OF HASHIMOTO'S THYROIDITIS



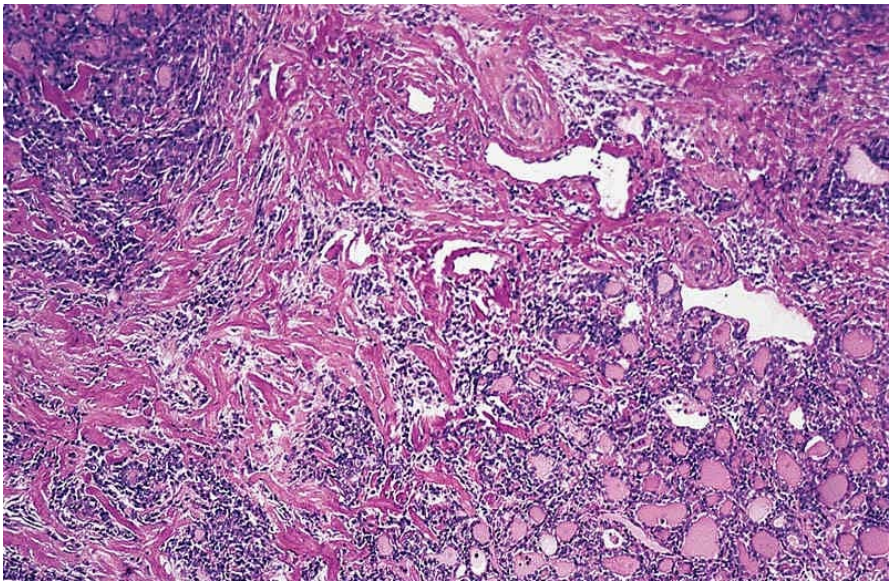
HISTOPATHOLOGY OF DEQUERVAIN'S THYROIDITIS



HISTOPATHOLOGY OF LYMPHOCYTIC THYROIDITIS



GROSS APPEARANCE OF REIDEL'S THYROIDITIS



HISTOPATHOLOGY OF REIDEL'S THYROIDITIS

A CASE OF DIFFUSE GOITRE

A CASE MULTI NODULAR GOITRE

A CASE OF HASHIMOTO'S THYRIDITIS

PRE OPERATIVE PICTURE

A CASE OF HASHIMOTO'S THYRIDITIS

POST OPERATIVE PICTURE

PROFORMA

Name : Age: Sex:

l.P.No. :

Address :

History

Chief Complaints :

Significant History :

Compressive Symptoms :

Examination

General Examination :

Vital Signs :

Signs of hyper/
Hypothyroidism :

Examination of the Neck :

Thyroid size :

Tenderness :

Consistency :

Nodularity :

Regional Nodes :

Other significant findings :

Other systemic examination :

CVS :

RS :

Abdomen :

CNS :

Clinical Diagnosis :

Investigations

1) Thyroid function tests :

T4 :

T3 :

TSH :

2) Anti- microsomal antibodies :

3) USG :

4) FNAC :

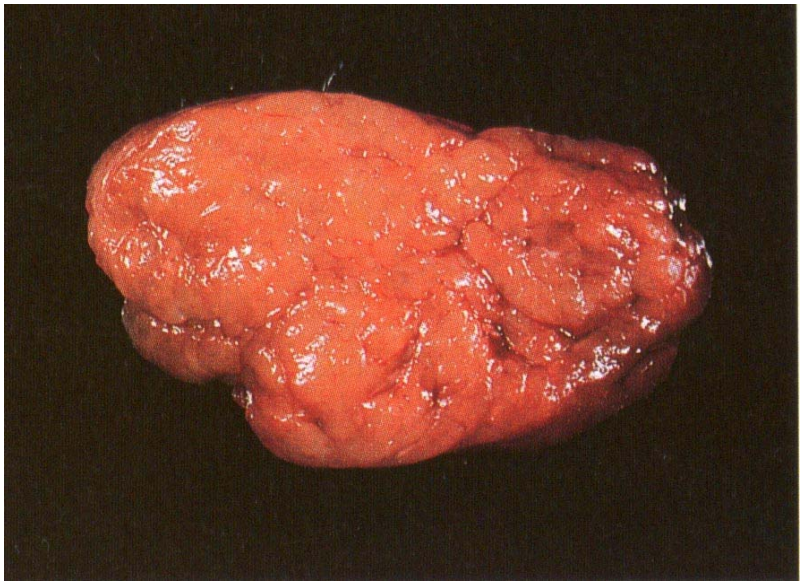
5) ESR :

Diagnosis

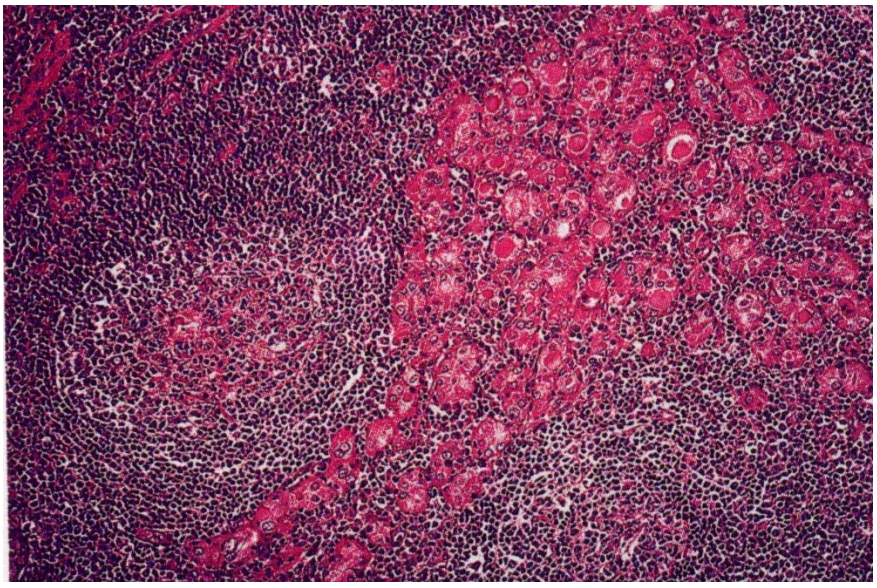
Operative Procedure (if done) :

Postoperative complications (if any) :

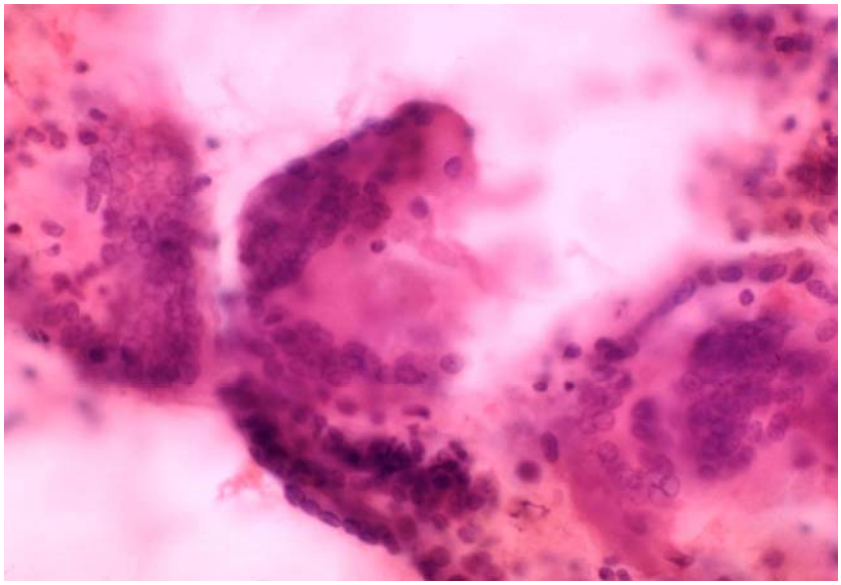
HPE report of Thyroidectomy Specimen :



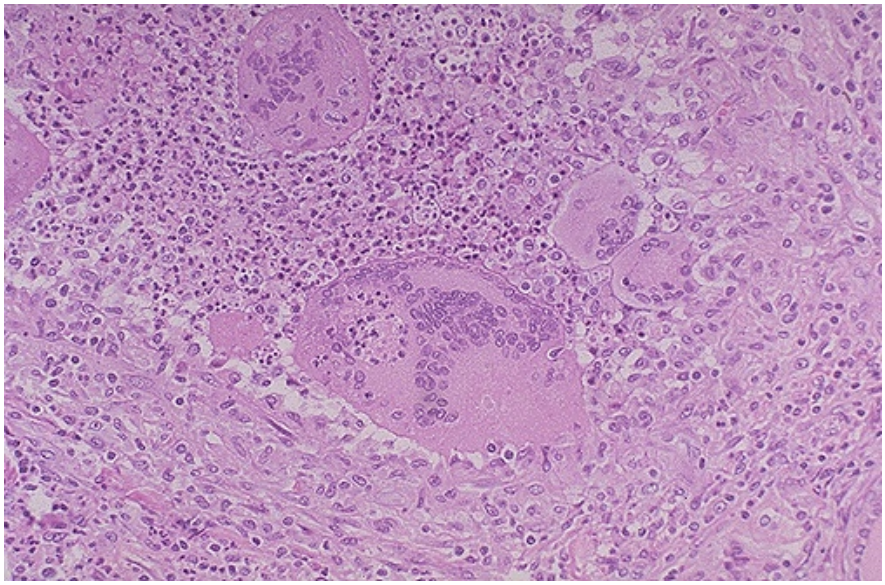
RESECTED SPECIMEN OF THYROID



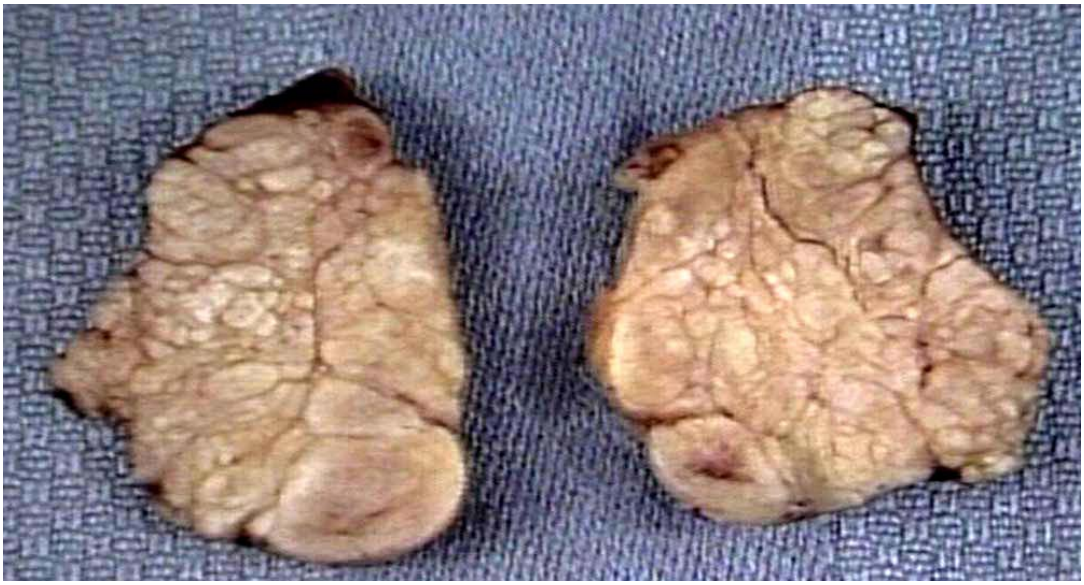
HISTOPATHOLOGY OF HASHIMOTO'S THYROIDITIS



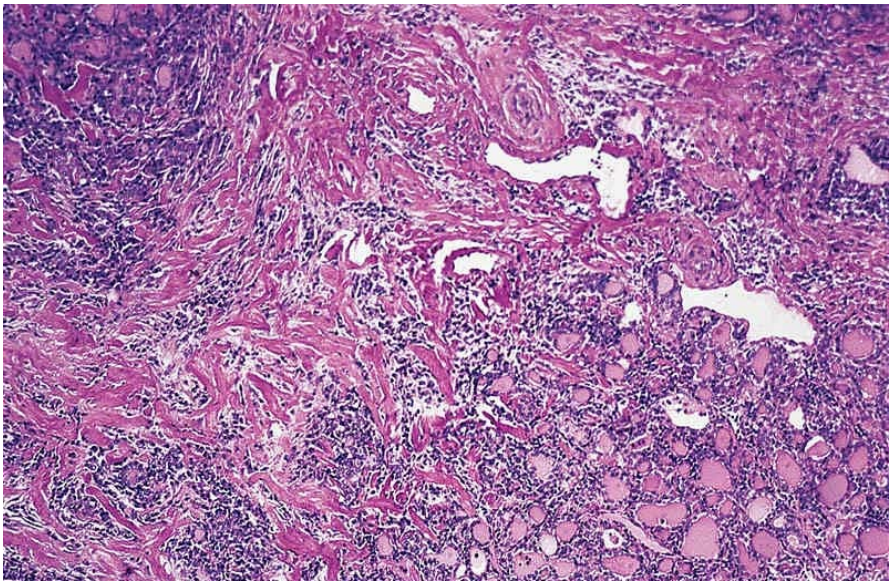
HISTOPATHOLOGY OF DEQUERVAIN'S THYROIDITIS



HISTOPATHOLOGY OF LYMPHOCYTIC THYROIDITIS



GROSS APPEARANCE OF REIDEL'S THYROIDITIS



HISTOPATHOLOGY OF REIDEL'S THYROIDITIS

A CASE OF DIFFUSE GOITRE

A CASE MULTI NODULAR GOITRE

A CASE OF HASHIMOTO'S THYRIDITIS

PRE OPERATIVE PICTURE

A CASE OF HASHIMOTO'S THYRIDITIS

POST OPERATIVE PICTURE

MASTER CHART

S.No.	NAME	IP NO	AGE	SEX	PRESENTATION	CLINICAL FEATURES	CYTOLOGICAL DIAGNOSIS	THYROID STATUS	RX
1	VIJAYA	60/08	35	F	M	S	CO	EU	NTT
2	RAJIMA	17/08	60	F	M	S	CO	EU	NTT
3	MANJULA	31/07	55	F	M	S	CO	EU	NTT
4	MAHAR	140/06	24	F	M	S	CO	EU	NTT
5	UMADEVI	53/07	40	F	S	S	CO	EU	HT
6	MALIKA	36/07	45	F	M	S	CO	EU	NTT
7	LAKSHMI	69/07	24	F	M	S	CO	EU	NTT
8	VANILA	105/7	27	F	M	C	CO	HO	Med
9	THIRUPESWARI	92/9	36	F	M	S	CO	EU	NTT
10	PUSHPAVALLI	1115/09	35	F	M	S	CO	EU	NTT
11	SUNDARI	332/05	40	F	M	S	CO	EU	NTT
12	VASUKI	73/09	27	F	M	S	CO	EU	NTT
13	DAVID	131/07	26	M	M	S	CA	EU	NTT
14	PONDIAMMAL	173/006	49	F	M	S	CO	EU	NTT
15	DEIVANI	281/7	25	F	M	S	CO	EU	NTT
16	LAKSHMI	83/4	39	F	M	S	CO	EU	NTT
17	MADTHU	183/07	40	F	M	S	CO	HF	NTT
18	NELLAMAL	83/9	45	F	D	C	CO	EU	NTT
19	BOOPATHI	54/9	34	F	M	S	CA	EU	NTT
20	DHANALAKSHMI	14/09	37	F	M	S	CO	EU	NTT
21	MALLIKA	58/09	30	F	M	S	CO	EU	NTT
22	LAKSHMI	34/09	40	F	M	S	CO	EU	NTT
23	SELVAMUTHU	7/09	34	F	M	S	CO	EU	NTT
24	KAVITH	140/09	19	F	M	S	CO	EU	HT
25	AVALLI	129/07	25	F	M	S	CO	EU	NTT
26	SUNDARI	34/07	69	F	M	S	CO	EU	NTT
27	KALA	107/08	40	F	M	S	CA	EU	NTT
28	PUSPHALATHA	173/09	49	F	M	S	CO	EU	NTT
29	SARATHA	353/07	30	F	M	S	CO	EU	NTT
30	POONGODI	175/05	28	F	M	S	CO	HO	Med
31	SUMATHI	88/09	36	F	M	S	CO	EU	NTT
32	SAROJA	282/07	34	F	M	S	CO	EU	NTT
33	PANDIAMMAL	317/05	51	F	M	S	CO	HE	NTT
34	NAGAMMAL	311/05	32	F	M	S	CO	EU	NTT
35	VASUKI	265/05	16	F	S	S	CO	EU	NTT
36	ARUNA	512/05	40	F	M	S	CO	EU	HT
37	JEEVA	60/08	40	F	M	S	CO	EU	NTT
38	PANCHU	48/08	35	F	M	S	CA	EU	NTT
39	SUSEELA	60/09	36	F	M	S	CO	EU	NTT
40	SUBULAKSHMI	48/07	23	F	M	S	CO	EU	NTT

S.No.	NAME	IP NO	AGE	SEX	PRESENTATION	CLINICAL FEATURES	CYTOLOGICAL DIAGNOSIS	THYROID STATUS	RX
41	JOTHI	31/06	60	F	M	S	CO	EU	ITT
42	AKILANAM	55/6	39	F	M	S	CO	EU	NTT
43	RAJATHI	415/07	35	F	M	S	CO	EU	NTT
44	PAPPAN	28/09	35	F	S	S	CA	HO	Med
45	MAYAMMAL	128/09	34	F	M	C	CO	EU	NTT
46	YASIN	60/09	38	F	M	S	CO	EU	NTT
47	THAVAMANI	40/07	43	F	M	S	CO	EU	NTT
48	DEIVAMANI	100/07	43	F	M	S	CO	EU	NTT
49	DOORVAI	45/107	30	F	M	S	CO	EU	NTT
50	POOMADEVI	405/07	26	F	M	S	CO	EU	NTT
51	MANOHARAN	151/07	60	F	M	S	CO	EU	NTT
52	SARIBA	55/07	15	F	S	S	CO	EU	HT
53	VANITHA	91/09	45	F	M	S	CO	EU	NTT
54	RATHIRAM	102/09	22	F	M	S	CO	EU	NTT
55	DEVI	362/07	32	F	M	S	CO	HO	Med
56	MURUGESWARI	92/07	40	F	M	C	CA	EU	NTT
57	THENMOZHI	91/07	40	F	D	S	CO	EU	NTT
58	AKILANDESWARI	26/07	33	F	M	S	CO	EU	NTT
59	THERASCA	411/07	36	F	M	S	CO	EU	NTT
60	ROJA	25/07	50	F	D	S	CO	EU	NTT
61	PANDIAMMAL	192/08	34	F	M	S	CO	EU	NTT
62	KAMATCHI	142/07	60	F	M	S	CO	EU	ITT
63	CHITRADEVI	370/05	40	F	M	S	CO	EU	NTT
64	NITHYA	32/07	60	F	D	C	CO	EU	NTT
65	MEHAL	31/07	45	F	M	S	CO	EU	NTT
66	SUNDARAWATHI	9/07	38	F	M	S	CO	EU	NTT
67	GOMATHY	569/05	36	F	M	S	CA	EU	NTT
68	USHA	105/07	26	F	M	S	CO	HO	Med
69	LAKSHI	100/08	20	F	M	S	CO	EU	NTT
70	MURUGESWARI	183/07	32	F	M	S	CO	EU	NTT
71	VASANTHA	148/08	40	F	M	S	CO	EU	NTT
72	LATHA	196/08	30	F	M	S	CO	EU	NTT
73	KAVITHA	125/07	24	F	D	S	CO	EU	NTT
74	MANOHAR	415/07	38	F	M	S	CO	EU	NTT
75	NATARAJAN	353/07	44	F	M	P	CO	EU	NTT
76	RAJATHI	279/05	55	F	M	S	CO	EU	NTT
77	VASANTHI	45/07	63	F	S	S	CA	EU	HT
78	SAVITHI	90/07	39	F	M	T	CO	EU	NTT
79	CHELLAM	105/08	42	F	M	S	CO	EU	NTT
80	KAMALAM	6/09	29	F	M	S	CO	EU	NTT
81	SUGANYA	140/09	35	F	M	S	CO	EU	NTT

S.No.	NAME	IP NO	AGE	SEX	PRESENTATION	CLINICAL FEATURES	CYTOLOGICAL DIAGNOSIS	THYROID STATUS	RX
82	PANDIAMMAL	32/09	70	F	M	S	CO	EU	NTT
83	ASTALAKSHMI	27/07	40	F	M	S	CO	EU	NTT
84	PADMA	122/09	26	F	S	S	CO	EU	NTT
85	SELVI	470/09	45	F	M	S	CO	EU	NTT
86	RANI	58/09	40	F	S	S	CO	EU	HT
87	JEYALAKSHMI	129/09	64	F	M	S	CO	EU	NTT
88	PERIYAMATCHI	62/03	47	F	M	S	CO	EU	NTT
89	KAVITH	102/01	62	F	M	S	CO	EU	NTT
90	PETCHI	126/09	29	F	M	C	CO	EU	NTT
91	KRISHNAVENI	151/09	40	F	M	S	CO	HO	Med
92	VANITHA	87/09	49	F	M	S	CO	EU	NTT
93	SARATHA	32/07	60	F	D	S	CA	EU	NTT
94	SEENIAMMAL	60/07	38	F	M	S	CO	EU	NTT
95	SUNDERESWARI	84/08	35	F	M	S	CO	EU	NTT
96	SAROJA	367/08	43	F	M	T	CO	EU	NTT
97	VAYAPOON	405/07	30	F	S	S	CO	EU	NTT
98	PORNAM	145/07	34	F	M	S	CO	EU	NTT
99	ANANTHAM	62/07	52	F	M	S	CA	EU	NTT
100	DHAVAMANI	72/08	34	F	M	S	CO	EU	NTT
101	MEENA	140/07	32	F	M	S	H	EU	Med
102	AMUTHA	80/08	34	F	D	S/D	H	EU	Med
103	THIRUPURANI	50/07	38	F	M	S	H	HO	Med
104	GOMATHI	183/07	22	F	M	S	H	EU	Med
105	NATARAJ AMMAL	37/07	34	F	M	S	H	EU	Med
106	MARI	55/07	36	F	M	S/P	H	EU	Med
107	JEGATHA	543/08	42	F	D	S	H	HO	Med
108	SUSEELA	529/07	35	F	M	S	H	EU	Med
109	NATHIYA	527/08	18	F	M	S/D	C	HO	NTT
110	KALEESWARI	569/08	30	F	S	S	H	EU	Med
111	KAMARNISHA	126/08	44	F	M	S	D	EU	Med
112	CHANDRAN	439/07	36	F	M	IP	H	EU	Med
113	KAVITHA	455/08	37	F	M	S	H	EU	Med
114	SUNDARAI	138/08	24	F	M	S	H	HE	Med
115	MUTHULAKSHMI	279/08	39	F	D	S	H	EU	Med
116	POONGODI	173/07	46	F	M	ID	H	HO	Med
117	KAVEN	193/07	26	F	M	S	L	EU	Med
118	MUNEESWARI	192/08	32	F	M	S	H	HO	Med
119	VAYAPURI	131/07	62	F	M	S	CO	EU	NTT
120	KAMATCHI	27/07	30	F	M	S	CO	EU	NTT
121	VELLIMANI	17/09	40	F	M	S	CO	EU	NTT
122	PANCHU	18/09	34	F	M	S	CO	EU	NTT

S.No.	NAME	IP NO	AGE	SEX	PRESENTATION	CLINICAL FEATURES	CYTOLOGICAL DIAGNOSIS	THYROID STATUS	RX
123	MEENA	140/09	35	F	M	S	CO	EU	NTT
124	MUTHULAKSHMI	173/09	46	F	M	C	CA	EU	NTT
125	VALLI	6-Feb	32	F	D	S	CO	EU	NTT
126	RANI	7-Jul	32	F	M	S	CO	EU	NTT
127	MURUGESWARI	100/07	25	F	M	S	CO	HO	Med
128	POOMADEVI	9-Dec	40	F	M	S	CO	EU	NTT
129	CHANDRA	15/09	17	F	M	S	CO	EU	NTT
130	FATHIMA	332/08	27	F	M	S	CO	EU	NTT
131	ANANTHAVALLI	187/9	32	F	M	S	CO	EU	NTT
132	RATHINAM	526/08	35	F	D	S	CO	EU	NTT
133	ALAGUPONU	69/07	20	F	M	S	CO	EU	NTT
134	MARY	362/05	40	F	M	C	CA	EU	NTT
135	SARASWATHI	141/09	40	F	S	S	CO	EU	NTT
136	MARAGATHAM	17/02	38	F	M	S	CO	EU	NTT
137	JEEVA	43/07	45	F	M	P	CO	EU	NTT
138	SUDHA	42/08	57	F	M	S	CO	EU	NTT
139	KAVITHA	10/09	29	F	M	S	CO	EU	NTT
140	MUTHULAKSHMI	9/05	45	F	M	S	CO	EU	NTT
141	MARY	17/05	37	F	M	S	CO	EU	NTT
142	SARATHA	570/09	30	F	S	P	CA	EU	HT
143	KAMATCHI	62/07	35	F	M	S	CO	EU	NTT
144	USHA	31/08	69	F	M	S	CO	EU	NTT
145	SANTHI	102/05	32	F	M	S	CO	EU	NTT
146	LAKSHMI	473/05	40	F	M	S	CO	EU	NTT
147	KAMALAM	122/05	28	F	M	S	CO	EU	NTT
148	MANOHARAN	124/07	45	M	S	S	CO	EU	NTT
149	VALLI	475/07	35	F	M	S	CO	EU	NTT
150	PARVATHI	45/08	40	F	M	P	CO	EU	NTT
151	DEVI	2/09	40	F	M	S	CO	EU	NTT
152	SELVI	328/09	57	F	M	S	CO	EU	NTT
153	MUTHUTHURAI	235/02	43	M	D	S	CO	EU	NTT
154	CHITRA	40/08	28	F	M	S	CA	EU	NTT
155	DILSITH	490/07	40	F	M	S	CO	EU	NTT
156	PALANIAMMAL	70/09	23	F	M	S	CO	EU	NTT
157	VISALATCHI	405/07	28	F	M	S	CO	EU	NTT
158	KAVERI	557/05	39	F	M	S	CO	EU	NTT
159	RAMALAKSHMI	8/05	36	F	M	S	CO	EU	NTT
160	ANITHA	143/09	40	F	M	S	CO	EU	NTT
161	MUTHU	126/07	43	F	M	S	CO	EU	NTT
162	EASWARI	527/07	30	F	S	S	CO	EU	NTT
163	USHA	187/03	60	F	M	S	CA	EU	NTT

S.No.	NAME	IP NO	AGE	SEX	PRESENTATION	CLINICAL FEATURES	CYTOLOGICAL DIAGNOSIS	THYROID STATUS	RX
164	PARVEEN	62/07	68	F	M	S	CO	EU	NTT
165	VEERAMMAL	108/07	35	F	M	S	CO	EU	NTT
166	PANDESSWARI	132/08	42	F	M	C	CO	EU	NTT
167	SIVARANJANI	328/09	50	F	M	S	CO	EU	NTT
168	RAJAMANI	156/07	26	F	M	S	CO	HE	NTT
169	SAVITHIRI	235/07	36	F	M	S	CO	EU	NTT
170	MADAVI	260/08	40	F	M	S	CO	EU	NTT
171	VALLI	367/09	46	F	M	S	CO	EU	NTT
172	RATHINAM	132/07	37	F	M	S	CO	EU	NTT
173	NAGAMMAL	127/09	40	F	S	S	CO	EU	NTT
174	KANIKA	148/07	32	F	M	S	CO	EU	NTT
175	KAMALAM	101/07	39	F	M	C	CO	EU	NTT
176	MARIMUTHU	9/07	27	F	M	S	CO	EU	NTT
177	PAVOON	349/05	45	F	M	S	CO	EU	NTT
178	MOOKAYEE	128/07	50	F	M	S	CO	EU	NTT
179	GAYATHIRI	517/05	37	F	M	S	H	HO	Med
180	VASANTHI	151/05	39	F	M	D	C	EU	NTT
181	REEKA	131/09	28	F	S	S	H	HO	Med
182	KARTHIKA	43/09	31	F	M	S	H	EU	Med
183	MUTHULAKSHMI	520/08	56	F	D	S	C	HO	NTT
184	MARIAMMAL	7/07	33	F	M	S	H	EU	Med
185	LATHA	28/07	49	F	M	S	H	EU	Med
186	NAGAVALLI	285/05	34	F	M	S	H	HO	Med
187	CHANDRA	111/08	43	F	M	S	CO	EU	NTT
188	SUDHA	62/08	27	F	S	S	CA	EU	NTT
189	VELMANI	30/07	30	F	S	S	CO	EU	NTT
190	PALANIAMMAL	22/08	35	F	S	S	CO	EU	NTT
191	RUKKUMANI	40/08	36	F	M	S	CO	EU	NTT
192	SELUBUMSELVI	80/06	35	F	M	S	CO	EU	NTT
193	KAMATCHI	527/08	60	F	M	S	CO	EU	NTT
194	NAGAMMAL	560/07	27	F	M	S	CO	EU	NTT
195	PRIYA	9/06	47	F	M	S	CO	EU	NTT
196	PALAMAL	145/08	35	F	M	S	CA	EU	NTT
197	MUTHULAXMI	148/08	30	F	D	D	CO	EU	NTT
198	USHA	62/07	69	F	M	S	CO	EU	NTT
199	SHANTHI	102/07	40	F	M	S	CO	EU	NTT
200	JEEVA	28/07	20	F	M	S	CO	EU	NTT
201	SALIMA	459/08	40	F	S	S	CO	EU	NTT
202	KAMALAM	347/07	37	F	S	S	CO	EU	NTT
203	SELVI	40/08	50	F	M	S	CO	EU	NTT
204	CHITRA	73/05	23	F	M	S	CO	EU	NTT

S.No.	NAME	IP NO	AGE	SEX	PRESENTATION	CLINICAL FEATURES	CYTOLOGICAL DIAGNOSIS	THYROID STATUS	RX
205	VASANTHI	40/07	32	F	M	S	CO	EU	NTT
206	CHELLAMA	6/08	29	F	M	S	CO	EU	NTT
207	VIMALA	33/05	55	F	S	S	CO	EU	HT
208	MOHAN	37/07	45	F	M	S	CO	EU	NTT
209	SUTHIRADEVI	68022	34	F	M	S	CO	EU	NTT
210	JEYACHITRA	46322	32	F	M	S	CO	EU	NTT
211	MARIYA	101913	37	F	M	S	CO	EU	NTT
212	SUBBULAKSHMI	13225	24	F	D	S	CO	HO	Med
213	SANTRA	72310	32	F	M	C	CO	EU	NTT
214	SULTA	150/01	40	F	M	S	CO	EU	NTT
215	DURGA	26686	32	F	M	S	CA	EU	NTT
216	PADMA	43592	67	F	M	S	CO	EU	NTT
217	PREETHI	13592	46	F	M	S	CO	EU	NTT
218	SANTHI	268948	43	F	M	S	CO	EU	NTT
219	MEENATCHI	89/98	40	F	S	S	CO	EU	NTT
220	LAKSHMI	4756	24	F	M	S	CO	EU	Med
221	PREMA	18032	35	F	M	S	CO	EU	NTT
222	MUNEESWARI	1349	60	F	M	S	CO	HO	NTT
223	KALEESWARI	2109	44	F	M	S	CO	EU	NTT
224	JEYALAKSHMI	3707	49	F	M	S	CA	EU	NTT
225	ROHINI	75451	34	F	S	S	CO	EU	NTT
226	SHANTHI	14223	39	F	M	S	CO	EU	NTT
227	PETCHIAMMAL	182/09	32	F	M	M	CO	EU	NTT
228	MUNIYASAMY	183/09	44	M	M	S	CO	EU	NTT
229	VANESWARI	41929	63	F	M	S	CA	EU	NTT
230	HASEENA	367/07	51	F	M	S	CO	EU	NTT
231	MUTHURANI	128/07	34	F	M	S	CO	EU	NTT
232	SAKUNTHALA	421/08	36	F	M	S	CO	EU	NTT
233	VIJAYA	311/08	35	F	M	S	CO	EU	NTT
234	VELAMMAL	70/07	47	F	M	S	CO	EU	NTT

ABBREVIATIONS

PRESENTATION : M - Multi nodular goitre ; D - Diffuse goitre ; S - Solitary Nodular goitre
CLINICAL FEATURES : S - Swelling ; P - Pain ; D - Dysphagia
CYTOLOGICAL DIAGNOSIS : H - Hashimoto's ; L - Lymphocytic ; D - Dequervain ;
CO - Colloid ; CA - Carcinoma
THYROID STATUS : EU - Euthyroid ; HO - Hypothyroid ; HE - Hyperthyroid
Rx : - NTT - Near Total Thyroidectomy ; HT - Hemi thyroidectomy

REFERENCES

1. Diagnostic endocrinology- 2nd edition- by W.Tabb Moore and Richard C. Eastman.
2. The medical clinics of North America- Thyroid diseases Jan 1991- Francis S.Greenspan (pages 61-78)
3. Oxford textbook of surgery-2 edition- Sir Peter J.Morris, William C.Wood.(page 1096 to 1120).
4. Ackerman's surgical pathology- 5th edition.
5. Silverberg' surgical pathology- 3 edition.
6. Diagnostic ultrasound- Rumack- 2nd edition (pages 703- 716).
7. Textbook of surgery- David C.Sabiston — 16th edition
8. Principles of surgery- 7th edition- Seymour LSchwartz (page 1661 to 1694).
9. Bailey & Love's short practice of surgery- 24th edition.
10. Essential surgical practice- Sir Alfred Cuschieri- 4th edition.
11. Harrison's principles of internal medicine- 15Ih edition (page 2060 to 2084).
12. Textbook of surgery- the Association of Surgeons of India- Dr.Ahamed Ali.(pages 709-723).

13. Davidson’s principles and practice of medicine — 19th edition (pages 686 to 702).
14. Medical endocrinology- 3rd edition.
15. The New Aird’s companion in surgical studies- Kevin G.l3umand, Anthony E.Young.
16. www.endocrineweb.com
17. www.health.a1lrefer.com
18. www.emedicine.com
19. www.medpix.com
20. www.patients.uptodate.com